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NEWS 5	Apr 19	US Patent Applications available in IFICDB, IFIPAT, and IFIUDB
NEWS 6	Apr 22	Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
NEWS 7	Apr 22	BIOSIS Gene Names now available in TOXCENTER
NEWS 8	Apr 22	Federal Research in Progress (FEDRIP) now available
NEWS 9	Jun 03	New e-mail delivery for search results now available
NEWS 10	Jun 10	MEDLINE Reload
NEWS 11	Jun 10	PCTFULL has been reloaded
NEWS 12	Jul 02	FOREGE no longer contains STANDARDS file segment
NEWS 13	Jul 22	USAN to be reloaded July 28, 2002; saved answer sets no longer valid
NEWS 14	Jul 29	Enhanced polymer searching in REGISTRY
NEWS 15	Jul 30	NETFIRST to be removed from STN
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NEWS 17	Aug 08	PHARMAMarketLetter(PHARMAML) - new on STN
NEWS 18	Aug 08	NTIS has been reloaded and enhanced
NEWS 19	Aug 09	JAPIO to be reloaded August 25, 2002
NEWS 20	Aug 19	Aquatic Toxicity Information Retrieval (AQUIRE) now available on STN
NEWS 21	Aug 19	IFIPAT, IFICDB, and IFIUDB have been reloaded
NEWS 22	Aug 19	The MEDLINE file segment of TOXCENTER has been reloaded
NEWS 23	Aug 26	Sequence searching in REGISTRY enhanced

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                                                           ENTRY    SESSION
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CA INDEXING COPYRIGHT (C) 2002 AMERICAN CHEMICAL SOCIETY (ACS)

=> e collier r john/au
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E3         277 --> COLLIER R JOHN/AU
E4          12     COLLIER R K/AU
E5           2     COLLIER R K JR/AU
E6           4     COLLIER R KIRK/AU
E7          18     COLLIER R L/AU
E8           1     COLLIER R L U/AU
E9           4     COLLIER R M/AU
E10          6     COLLIER R N/AU
E11          3     COLLIER R O/AU
E12          5     COLLIER R O JR/AU

=> s e1-e3
L1         293 ("COLLIER R J *"/AU OR "COLLIER R J JR"/AU OR "COLLIER R JOHN"/A
               U)
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=> e bradley kenneth a/au
E1            4      BRADLEY KEN C/AU
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E12           1      BRADLEY KENT P/AU

=> s e2-e3
L2           11 ("BRADLEY KENNETH"/AU OR "BRADLEY KENNETH A"/AU)

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=> s e3
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=> s e3
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=> e young johna t/au
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E12          11      YOUNG JONATHAN D/AU

=> e young john a t/au
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=> s e3
L6           76 "YOUNG JOHN A T"/AU

=> e youngj a t/au

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E12           2     YOUNGJOHN N R/AU

=> e young j a t/au
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E12           3     YOUNG J C F/AU

=> s e3-e4
L7           99 ("YOUNG J A T"/AU OR "YOUNG J A T *"/AU)

=> d his

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     FILE 'BIOSIS, MEDLINE, AGRICOLA, EMBASE, CABA, WPIDS, JAPIO, BIOTECHDS,
     LIFESCI, CAPLUS, USPATFULL, USPAT2' ENTERED AT 16:15:12 ON 29 AUG 2002
                 E COLLIER R JOHN/AU
L1           293 S E1-E3
                 E BRADLEY KENNETH A/AU
L2            11 S E2-E3
                 E BRADLEY K A/AU
L3           257 S E2-E3
                 E MOGRIDGE JEREMY/AU
L4            30 S E3
                 E MORGRIDGE J/AU
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L5            50 S E3
                 E YOUNG JOHNA T/AU
                 E YOUNG JOHN A T/AU
L6            76 S E3
                 E YOUNGJ A T/AU
                 E YOUNG J A T/AU
L7            99 S E3-E4

=> s l1-l7
L8           785 (L1 OR L2 OR L3 OR L4 OR L5 OR L6 OR L7)

=> s l8 and anthra?
L9           117 L8 AND ANTHRA?

=> s l9 and protective antigen
L10           92 L9 AND PROTECTIVE ANTIGEN

=> dup rem l10
PROCESSING COMPLETED FOR L10
L11           44 DUP REM L10 (48 DUPLICATES REMOVED)

=> d bib ab 1-44

L11  ANSWER 1 OF 44  CAPLUS  COPYRIGHT 2002 ACS        DUPLICATE 1
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AN 2002:256744 CAPLUS
DN 136:299672
TI Sequences of the mutant **anthrax** toxin **protective antigen** (PA) and uses thereof as vaccine for the treatment and prevention of bacterial infection
IN **Collier, R. John**; Sellman, Bret R.
PA USA
SO U.S. Pat. Appl. Publ., 37 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2002039588	A1	20020404	US 2001-848909	20010504
PRAI US 2000-201800P	P	20000504		

AB The invention provides sequences of eighteen mutant forms of pore-forming toxins, in particular, mutants of **anthrax** toxin **protective antigen** (PA). These mutant toxins may be used in vaccines for the prevention of bacterial infection. Addnl., dominant neg. mutants may be administered as therapeutics for the treatment of bacterial infection.

L11 ANSWER 2 OF 44 CAPLUS COPYRIGHT 2002 ACS
AN 2002:449716 CAPLUS
DN 137:29035
TI Sequences of a human receptor for B. **anthracis** toxin and therapeutical uses
IN **Young, John A. T.**; **Bradley, Kenneth A.**; Collier, Robert J.; Mogridge, Jeremy S.
PA Wisconsin Alumni Research Foundation, USA
SO PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002046228	A2	20020613	WO 2001-US30941	20011003

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2000-251481P P 20001205

AB The present invention discloses sequences of a human receptor for B. **anthracis** toxin and its therapeutical uses. Specifically, the present invention relates to a human **anthrax** toxin receptor and polynucleotides encoding the receptor as well as related proteins and polynucleotides, vectors contg. the polynucleotides and proteins, host cells contg. related polynucleotide mols., and cells displaying no **anthrax** toxin receptor on an exterior surface of the cells. The present invention also relates to methods for identifying mols. that bind the **anthrax** toxin receptor and mols. that reduce the toxicity of **anthrax** toxin. Finally, the present invention provides methods for treating human and non-human animals suffering from **anthrax**.

L11 ANSWER 3 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE

2
AN 2002:340528 BIOSIS
DN PREV200200340528
TI Mapping the lethal factor and edema factor binding sites on oligomeric **anthrax protective antigen**.
AU Cunningham, Kristina; Lacy, D. Borden; **Mogridge, Jeremy; Collier, R. John (1)**
CS (1) Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA, 02115: jcollier@hms.harvard.edu USA
SO Proceedings of the National Academy of Sciences of the United States of America, (May 14, 2002) Vol. 99, No. 10, pp. 7049-7053. http://www.pnas.org. print.
ISSN: 0027-8424.
DT Article
LA English
AB Assembly of **anthrax** toxin complexes at the mammalian cell surface involves competitive binding of the edema factor (EF) and lethal factor (LF) to heptameric oligomers and lower order intermediates of PA63, the activated carboxyl-terminal 63-kDa fragment of **protective antigen** (PA). We used sequence differences between PA63 and homologous PA-like proteins to delineate a region within domain 1' of PA that may represent the binding site for these ligands. Substitution of alanine for any of seven residues in or near this region (R178, K197, R200, P205, I207, I210, and K214) strongly inhibited ligand binding. Selected mutations from this set were introduced into two oligomerization-deficient PA mutants, and the mutants were used in various combinations to map the single ligand site within dimeric PA63. The site was found to span the interface between two adjacent subunits, explaining the dependence of ligand binding on PA oligomerization. The locations of residues comprising the site suggest that a single ligand molecule sterically occludes two adjacent sites, consistent with the finding that the PA63 heptamer binds a maximum of three ligand molecules. These results elucidate the process by which the components of **anthrax** toxin, and perhaps other binary bacterial toxins, assemble into toxic complexes.

L11 ANSWER 4 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 3
AN 2002:340526 BIOSIS
DN PREV200200340526
TI The lethal and edema factors of **anthrax** toxin bind only to oligomeric forms of the **protective antigen**.
AU **Mogridge, Jeremy**; Cunningham, Kristina; Lacy, D. Borden; Mourez, Michael; **Collier, R. John (1)**
CS (1) Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA, 02115: jcollier@hms.harvard.edu USA
SO Proceedings of the National Academy of Sciences of the United States of America, (May 14, 2002) Vol. 99, No. 10, pp. 7045-7048. http://www.pnas.org. print.
ISSN: 0027-8424.
DT Article
LA English
AB The three proteins that comprise **anthrax** toxin, edema factor (EF), lethal factor (LF), and **protective antigen** (PA), assemble at the mammalian cell surface into toxic complexes. After binding to its receptor, PA is proteolytically activated, yielding a carboxyl-terminal 63-kDa fragment (PA63) that coordinates assembly of the complexes, promotes their endocytosis, and translocates EF and LF to the cytosol. PA63 spontaneously oligomerizes to form symmetric ring-shaped heptamers that are capable of binding three molecules of EF and/or LF as competing ligands. To determine whether binding of these ligands depends on oligomerization of PA63, we prepared two oligomerization-deficient forms of this protein, each mutated on a different PA63-PA63 contact face.

gtoreq12 ANG. The channels are presumed to be heptameric "mushrooms", with an extracellular "cap" region and a membrane-inserted, beta-barrel "stem". Although the crystal structure of the water-soluble monomeric form has been resolved to 2.1 ANG and that of the heptameric "prepore" to 4.5 ANG, the structure for the membrane-bound channel (pore) has not been determined. We have engineered mutant channels that are cysteine-substituted in residues in the putative beta-barrel, and identified the residues lining the channel lumen by their accessibility to a water-soluble sulfhydryl-specific reagent. The reaction with lumen-exposed cysteinyl side chains causes a drop in channel conductance, which we used to map the residues that line the pore. Our results indicate that the beta-barrel structure extends beyond the bilayer and involves residues that are buried in the monomer. The implication is that major rearrangement of domains in the prepore cap region is required for membrane insertion of the beta-barrel stem.

L11 ANSWER 7 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 6
AN 2002:179668 BIOSIS
DN PREV200200179668
TI Stoichiometry of **anthrax** toxin complexes.
AU **Mogridge, Jeremy**; Cunningham, Kristina; **Collier, R. John (1)**
CS (1) Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA, 02115: jcollier@hms.harvard.edu USA
SO Biochemistry, (January 22, 2002) Vol. 41, No. 3, pp. 1079-1082. http://pubs.acs.org/journals/bichaw/. print.
ISSN: 0006-2960.
DT Article
LA English
AB After being proteolytically activated, the **protective antigen** (PA) moiety of **anthrax** toxin self-associates to form symmetric, ring-shaped heptamers. Heptameric PA competitively binds the enzymatic moieties of the toxin, edema factor and lethal factor, and translocates them across the endosomal membrane by a pH-dependent process. We used two independent approaches to determine how many of the seven identical EF/LF binding sites of the PA heptamer can be occupied simultaneously. We measured isotope ratios in complexes assembled from differentially radiolabeled toxin subunits, and we determined the molecular masses of unlabeled complexes by multiangle laser light scattering. Both approaches yielded the same value: the PA heptamer in solution binds three molecules of protein ligand under saturating conditions. This suggests that each bound ligand sterically occludes the binding sites of two PA subunits. According to this model, a ligand-saturated heptamer is asymmetric, with the sites of six of the seven subunits occluded. These results contribute to the conceptual framework for understanding the mechanism of membrane translocation by **anthrax** toxin.

L11 ANSWER 8 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2002:353105 BIOSIS
DN PREV200200353105
TI Fluorescence studies on spatial relations between **anthrax** lethal toxin components.
AU Croney, John C. (1); Cunningham, Kristina M.; **Collier, R. John**; Jameson, David M. (1)
CS (1) University of Hawaii, Honolulu, HI USA
SO Biophysical Journal, (January, 2002) Vol. 82, No. 1 Part 2, pp. 430a. http://intl.biophysj.org/. print.
Meeting Info.: 46th Annual Meeting of the Biophysical Society San Francisco, California, USA February 23-27, 2002
ISSN: 0006-3495.

SL English
AB The **protective antigen** (PA) moiety of **anthrax** toxin delivers the toxin's enzymatic moieties to the cytosol of mammalian cells by a mechanism associated with its ability to heptamerize and form a transmembrane pore. Here we report that mutations in Lys-397, Asp-425, or Phe-427 ablate killing of CHO-K1 cells by a cytotoxic PA ligand. These mutations blocked PA's ability to mediate pore formation and translocation in cells but had no effect on its receptor binding, proteolytic activation, or ability to oligomerize and bind the toxin's enzymatic moieties. The mutation-sensitive residues lie in the 2beta7-2beta8 and 2beta10-2beta11 loops of domain 2 and are distant both in primary structure and topography from the 2beta2-2beta3 loop, which is believed to participate in formation of a transmembrane beta-barrel. These results suggest that Lys-397, Asp-425, and Phe-427 participate in conformational rearrangements of a heptameric pore precursor that are necessary for pore formation and translocation. Identification of these residues will aid in elucidating the mechanism of translocation and may be useful in developing therapeutic and prophylactic agents against **anthrax**.

L11 ANSWER 12 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 8
AN 2001:157442 BIOSIS
DN PREV200100157442
TI Involvement of domain 3 in oligomerization by the **protective antigen** moiety of **anthrax** toxin.
AU **Mogridge, Jeremy**; Mourez, Michael; **Collier, R. John (1)**
CS (1) Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA, 02115: jcollier@hms.harvard.edu USA
SO Journal of Bacteriology, (March, 2001) Vol. 183, No. 6, pp. 2111-2116. print.
ISSN: 0021-9193.
DT Article
LA English
SL English
AB **Protective antigen** (PA), a component of **anthrax** toxin, binds receptors on mammalian cells and is activated by a cell surface protease. The resulting active fragment, PA63, forms ring-shaped heptamers, binds the enzymic moieties of the toxin, and translocates them to the cytosol. Of the four crystallographic domains of PA, domain 1 has been implicated in binding the enzymic moieties; domain 2 is involved in membrane insertion and oligomerization; and domain 4 binds receptor. To determine the function of domain 3, we developed a screen that allowed us to isolate random mutations that cause defects in the activity of PA. We identified several mutations in domain 3 that affect monomer-monomer interactions in the PA63 heptamer, indicating that this may be the primary function of this domain.

L11 ANSWER 13 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 9
AN 2001:264011 BIOSIS
DN PREV200100264011
TI Dominant-negative mutants of a toxin subunit: An approach to therapy of **anthrax**.
AU Sellman, Bret R.; Mourez, Michael; **Collier, R. John (1)**
CS (1) Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA, 02115: jcollier@hms.harvard.edu USA
SO Science (Washington D C), (27 April, 2001) Vol. 292, No. 5517, pp. 695-697. print.
ISSN: 0036-8075.
DT Article
LA English
SL English

AB The **protective antigen** moiety of **anthrax** toxin translocates the toxin's enzymic moieties to the cytosol of mammalian cells by a mechanism that depends on its ability to heptamerize and insert into membranes. We identified dominant-negative mutants of **protective antigen** that co-assemble with the wild-type protein and block its ability to translocate the enzymic moieties across membranes. These mutants strongly inhibited toxin action in cell culture and in an animal intoxication model, suggesting that they could be useful in therapy of **anthrax**.

L11 ANSWER 14 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2001:138034 BIOSIS
DN PREV200100138034
TI Studies of **anthrax** toxin **Protective Antigen** oligomerization in solution using fluorescence polarization.
AU Gao-Sheridan, H. Samantha (1); Cunningham, Kristina M. (1); Jameson, David M.; **Collier, R. John (1)**
CS (1) Harvard Medical School, 200 Longwood Ave., Boston, MA, 02115 USA
SO Biophysical Journal, (January, 2001) Vol. 80, No. 1 Part 2, pp. 410a. print.
Meeting Info.: 45th Annual Meeting of the Biophysical Society Boston, Massachusetts, USA February 17-21, 2001 Biophysical Society . ISSN: 0006-3495.
DT Conference
LA English
SL English

L11 ANSWER 15 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2001:138033 BIOSIS
DN PREV200100138033
TI Fluorescence investigations into the assembly of **anthrax** lethal toxin.
AU Cunningham, Kristina M. (1); Gao-Sheridan, H. Samantha (1); Jameson, David M.; **Collier, R. John (1)**
CS (1) Harvard Medical School, 200 Longwood Avenue, Boston, MA, 02115 USA
SO Biophysical Journal, (January, 2001) Vol. 80, No. 1 Part 2, pp. 410a. print.
Meeting Info.: 45th Annual Meeting of the Biophysical Society Boston, Massachusetts, USA February 17-21, 2001 Biophysical Society . ISSN: 0006-3495.
DT Conference
LA English
SL English

L11 ANSWER 16 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 10
AN 2001:566771 BIOSIS
DN PREV200100566771
TI Crystal structure of the **anthrax** lethal factor.
AU Pannifer, Andrew D.; Wong, Thiang Yian; Schwarzenbacher, Robert; Renatus, Martin; Petosa, Carlo; Bienkowska, Jadwiga; Lacy, D. Borden; **Collier, R. John**; Park, Sukjoon; Leppla, Stephen H.; Hanna, Philip; Liddington, Robert C. (1)
CS (1) Burnham Institute, 10901 North Torrey Pines Road, La Jolla, CA, 92037: rlidding@burnham.org USA
SO Nature (London), (8 November, 2001) Vol. 414, No. 6860, pp. 229-233. print.
ISSN: 0028-0836.
DT Article
LA English
SL English
AB Lethal factor (LF) is a protein (relative molecular mass 90,000) that is

L11 ANSWER 20 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2000:489596 BIOSIS
DN PREV200000489717
TI Advances in understanding the structure and function of **anthrax protective antigen**.
AU Sellman, B. (1); **Mogridge, J. (1)**; Mourez, M. (1); Collier, R. J. (1)
CS (1) Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA USA
SO Medical Microbiology and Immunology, (September, 2000) Vol. 189, No. 1, pp. 47. print.
Meeting Info.: 4th International Workshop on Pore-Forming Toxins Trento, Italy September 14-17, 2000
ISSN: 0300-8584.
DT Conference
LA English
SL English

L11 ANSWER 21 OF 44 CAPLUS COPYRIGHT 2002 ACS
AN 1999:549282 CAPLUS
DN 131:166479
TI Inhibition of toxin translocation
IN **Collier, R. John**; Benson, Erika L.; Finkelstein, Alan
PA President and Fellows of Harvard College, USA
SO PCT Int. Appl., 36 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9942473	A1	19990826	WO 1999-US3457	19990218
	W: AU, CA, JP				
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	AU 9927710	A1	19990906	AU 1999-27710	19990218
PRAI	US 1998-75286P	P	19980218		
	WO 1999-US3457	W	19990218		

AB In general, the invention features a mutant pore-forming toxin, wherein the toxin comprises a mutation in an amino acid that forms the transmembrane pore of said toxin. Also included is substantially pure nucleic acid that encodes the mutant pore-forming toxin, as well as methods of decreasing toxicity of a pore-forming toxin by administering a mutant pore-forming toxin in a dose sufficient to inhibit translocation of a pore-forming toxin.
RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 22 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 13
AN 1999:417145 BIOSIS
DN PREV199900417145
TI **Anthrax protective antigen**: Prepore-to-pore conversion.
AU Miller, Carl J.; Elliott, Jennifer L.; **Collier, R. John (1)**
CS (1) 200 Longwood Ave., Boston, MA, 02115 USA
SO Biochemistry, (Aug. 10, 1999) Vol. 38, No. 32, pp. 10432-10441.
ISSN: 0006-2960.
DT Article
LA English
SL English
AB PA63, the active 63 kDa form of **anthrax protective**

antigen, forms a heptameric ring-shaped oligomer that is believed to represent a precursor of the membrane pore formed by this protein. When maintained at pH gtoreq8.0, this "prepore" dissociated to monomeric subunits upon treatment with SDS at room temperature, but treatment at pH ltoreq7 (or with beta-octylglucoside at pH 8.0) caused it to convert to an SDS-resistant pore-like form. Transition to this form involved major changes in the conformation of loop 2 of domain 2 (D2L2), as evidenced by (i) occlusion of a chymotrypsin site within D2L2 and (ii) excimer formation by pyrene groups linked to N306C within this loop. The pore-like form retained the capacity to bind **anthrax** toxin A moieties and cell surface receptors, but was unable to form pores in membranes or mediate translocation. Mutant PA63 in which D2L2 had been deleted was inactive in pore formation and translocation but, like the prepore, was capable of forming heptamers that converted to an SDS-resistant form under acidic conditions. Our findings support a model of pore formation in which the D2L2 loops move to the membrane-proximal face of the heptamer and interact to form a 14-strand transmembrane beta-barrel. Concomitantly, domain 2 undergoes a major conformational rearrangement, independent of D2L2, that renders the heptamer resistant to dissociation by SDS. These results provide a basis for further exploration of the role of PA63 in translocation of **anthrax** toxin's enzymic moieties across membranes.

L11 ANSWER 23 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 14
AN 1999:357546 BIOSIS
DN PREV199900357546
TI Cytotoxic T-lymphocyte epitopes fused to **anthrax** toxin induce protective antiviral immunity.
AU Doling, Amy M.; Ballard, Jimmy D.; Shen, Hao; Krishna, Kaja Murali; Ahmed, Rafi; **Collier, R. John**; Starnbach, Michael N. (1)
CS (1) Department of Microbiology and Molecular Genetics, Harvard Medical School, 200 Longwood Ave., Boston, MA, 02115 USA
SO Infection and Immunity, (July, 1999) Vol. 67, No. 7, pp. 3290-3296. ISSN: 0019-9567.
DT Article
LA English
SL English
AB We have investigated the use of the **protective antigen** (PA) and lethal factor (LF) components of **anthrax** toxin as a system for in vivo delivery of cytotoxic T-lymphocyte (CTL) epitopes. During intoxication, PA directs the translocation of LF into the cytoplasm of mammalian cells. Here we demonstrate that antiviral immunity can be induced in BALB/c mice immunized with PA plus a fusion protein containing the N-terminal 255 amino acids of LF (LFn) and an epitope from the nucleoprotein (NP) of lymphocytic choriomeningitis virus. We also demonstrate that BALB/c mice immunized with a single LFn fusion protein containing NP and listeriolysin O protein epitopes in tandem mount a CTL response against both pathogens. Furthermore, we show that NP-specific CTL are primed in both BALB/c and C57BL/6 mice when the mice are immunized with a single fusion containing two epitopes, one presented by Ld and one presented by Db. The data presented here demonstrate the versatility of the **anthrax** toxin delivery system and indicate that this system may be used as a general approach to vaccinate outbred populations against a variety of pathogens.

L11 ANSWER 24 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 15
AN 1999:338648 BIOSIS
DN PREV199900338648
TI **Anthrax** toxin entry into polarized epithelial cells.
AU Beauregard, Kathryn E.; Wimer-Mackin, Susan; **Collier, R. John**;

Lencer, Wayne I. (1)
CS (1) GI Cell Biology, Children's Hospital, 300 Longwood Ave., Enders 1220, Boston, MA, 02115 USA
SO Infection and Immunity, (June, 1999) Vol. 67, No. 6, pp. 3026-3030. ISSN: 0019-9567.
DT Article
LA English
SL English
AB We examined the entry of **anthrax** edema toxin (EdTx) into polarized human T84 epithelial cells using cyclic AMP-regulated Cl- secretion as an index of toxin entry. EdTx is a binary A/B toxin which self assembles at the cell surface from **anthrax** edema factor and **protective antigen** (PA). PA binds to cell surface receptors and delivers EF, an adenylate cyclase, to the cytosol. EdTx elicited a strong Cl- secretory response when it was applied to the basolateral surface of T84 cells but no response when it was applied to the apical surface. PA alone had no effect when it was applied to either surface. T84 cells exposed basolaterally bound at least 30-fold-more PA than did T84 cells exposed apically, indicating that the PA receptor is largely or completely restricted to the basolateral membrane of these cells. The PA receptor did not fractionate with detergent-insoluble caveola-like membranes as cholera toxin receptors do. These findings have implications regarding the nature of the PA receptor and confirm the view that EdTx and CT coopt fundamentally different subcellular systems to enter the cell and cause disease.

L11 ANSWER 25 OF 44 CAPLUS COPYRIGHT 2002 ACS
AN 2001:318089 CAPLUS
DN 135:225502
TI Pore formation by **anthrax protective antigen**
AU Benson, Ericka L.; Huynh, Paul D.; Finkelstein, Alan; **Collier, R. John**
CS Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA, 02115, USA
SO Microbial Ecology and Infectious Disease, [derived from Two International Meetings on Microbial Ecology and Infectious Disease], National Institute of Health, Bethesda, MD, United States, July, 1996 and Israel Center for Emerging Diseases, Ma'al Hachamish, Israel, Apr., 1998 (1999), 97-108. Editor(s): Rosenberg, Eugene. Publisher: ASM Press, Herndon, Va. CODEN: 69BGCS
DT Conference
LA English
AB The channel-lining residues of PA63 (63-kDa fragment of **protective antigen**) have been identified by observing the response to methanethiosulfonate ethyltrimethylammonium (MTS-ET) of channels contg. cysteine substitutions within a disordered, amphipathic loop (D2L2). The pattern of MTS-ET inhibition supports the model of insertion of each D2L2 as an antiparallel .beta.-hairpin, with alternating hydrophobic and hydrophilic residues lining the membrane and aq. pore, resp. Single-channel expts. showing multiple stepwise conductance changes following addn. of MTS-ET confirm that the PA63 channel is oligomeric. Taken together, the results support the model of pore formation of PA63 as a transmembrane .beta.-barrel formed from .beta.-hairpins contributed by each PA63 protomer.
RE.CNT 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 26 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 16
AN 1999:16701 BIOSIS
DN PREV199900016701
TI Characterization of membrane translocation by **anthrax**

protective antigen.

AU Wesche, Jorgen; Elliott, Jennifer L.; Falnes, Pal O.; Olsnes, Sjur; **Collier, R. John (1)**

CS (1) Dep. Microbiol. Mol. Genet., Harvard Med. Sch., 200 Longwood Ave., Boston, MA 02115 USA

SO Biochemistry, (Nov. 10, 1998) Vol. 37, No. 45, pp. 15737-15746. ISSN: 0006-2960.

DT Article

LA English

AB Solving the crystallographic structure of the ring-shaped heptamer formed by **protective antigen** (PA), the B moiety of **anthrax** toxin, has focused attention on understanding how this oligomer mediates membrane translocation of the toxin's A moieties. We have developed an assay for translocation in which radiolabeled ligands are bound to proteolytically activated PA (PA63) at the surface of CHO or L6 cells, and translocation across the plasma membrane is induced by lowering the pH. The cells are then treated with Pronase E to degrade residual surface-bound material, and protected ligands are quantified after fractionation by SDS-PAGE. Translocation was most efficient (35%-50%) with LFN, the N-terminal PA binding domain of the **anthrax** lethal factor (LF). Intact LF, edema factor (EF), or fusion proteins containing LFN fused to certain heterologous proteins (the diphtheria toxin A chain (DTA) or dihydrofolate reductase (DHFR)) were less efficiently translocated (15%-20%); and LFN fusions to several other proteins were not translocated at all. LFN with different N-terminal residues was found to be degraded according to the N-end rule by the proteasome, and translocation of LFN fused to a mutant form of DHFR with a low affinity for methotrexate (MTX) protected cells from the effects of MTX. Both results are consistent with a cytosolic location of protected proteins. Evidence that a protein must unfold to be translocated was obtained in experiments showing that (i) translocation of LFNDTA was blocked by introduction of an artificial disulfide into the DTA moiety, and (ii) translocation of LFNDHFR and LFNDTA was blocked by their ligands (MTX and adenine, respectively). These results demonstrate that the acid-induced translocation by **anthrax** toxin closely resembles that of diphtheria toxin, despite the fact that these two toxins are unrelated and form pores by different mechanisms.

L11 ANSWER 27 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 17

AN 1998:480596 BIOSIS

DN PREV199800480596

TI **Anthrax** toxin as a molecular tool for stimulation of cytotoxic T lymphocytes: Disulfide-linked epitopes, multiple injections, and role of CD4+ cells.

AU Ballard, Jimmy D.; **Collier, R. John**; Starnbach, Michael N. (1)

CS (1) Harvard Medical Sch., Dep. Microbiol. Mol. Genet., 200 Longwood Ave., Boston, MA 02115 USA

SO Infection and Immunity, (Oct., 1998) Vol. 66, No. 10, pp. 4696-4699. ISSN: 0019-9567.

DT Article

LA English

AB We have previously demonstrated that **anthrax** toxin-derived proteins, **protective antigen** (PA) and the amino-terminal portion of lethal factor (LFn), can be used in combination to deliver heterologous molecules to the cytosol of mammalian cells. In this study we examined the ability of an LFn-peptide disulfide-linked heterodimer to prime cytotoxic T lymphocytes (CTL) in the presence of PA. A mutant of LFn that contains a carboxy-terminal reactive cysteine was generated. This form of LFn could be oxidized with a synthetic cysteine containing peptide to form a heterodimer of the protein and peptide. Mice injected with the heterodimer plus PA mounted a peptide-specific CTL

cytotoxic T-lymphocyte epitope from ovalbumin.

AU Ballard, Jimmy D.; Doling, Amy M.; Beauregard, Kathryn; **Collier, R. John**; Starnbach, Michael N. (1)

CS (1) Dep. Microbiol. Molecular Genetics, Harvard Med. Sch., 200 Longwood Ave., Boston, MA 02115 USA

SO Infection and Immunity, (Feb., 1998) Vol. 66, No. 2, pp. 615-619. ISSN: 0019-9567.

DT Article

LA English

AB We reported earlier that a nontoxic form of **anthrax** toxin was capable of delivering a cytotoxic T-lymphocyte (CTL) epitope in vivo, such that a specific CTL response was primed against the epitope. The epitope, of bacteria) origin, was fused to an N-terminal fragment (LFn) from the lethal-factor component of the toxin, and the fusion protein was injected, together with the **protective antigen** (PA) component, into BALB/c mice. Here we report that PA plus LFn is capable of delivering a different epitope-OVA257-264 from ovalbumin. Delivery was accomplished in a different mouse haplotype, H-2Kb and occurred in vitro as well as in vivo. An OVA257-264-specific CTL clone, GA-4, recognized EL-4 cells treated in vitro with PA plus as little as 30 fmol of the LFn-OVA257-264 fusion protein. PA mutants attenuated in toxin self-assembly or translocation were inactive, implying that the role of PA in epitope delivery is the same as that in toxin action. Also, we showed that OVA257-264-specific CTL could be induced to proliferate by incubation with splenocytes treated with PA plus LFn-OVA257-264. These findings imply that PA-LFn may serve as a general delivery vehicle for CTL epitopes in vivo and as a safe, efficient tool for the ex vivo expansion of patient-derived CTL for use in adoptive immunotherapy.

L11 ANSWER 30 OF 44 CAPLUS COPYRIGHT 2002 ACS

AN 1997:503256 CAPLUS

DN 127:126641

TI Use of toxin peptides and/or affinity handles for the delivery of compounds into cells

IN **Collier, R. John**; Blanke, Steven R.; Milne, Jill C.; Lyszak, Ericka L.; Ballard, Jimmy D.; Starnbach, Michael N.

PA President and Fellows of Harvard College, USA; Collier, R. John; Blanke, Steven R.; Milne, Jill C.; Lyszak, Ericka L.; Ballard, Jimmy D.; Starnbach, Michael N.

SO PCT Int. Appl., 66 pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9723236	A1	19970703	WO 1996-US20463	19961213
	W: AU, CA, JP, US				
	RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	CA 2239909	AA	19970703	CA 1996-2239909	19961213
	AU 9722401	A1	19970717	AU 1997-22401	19961213
	AU 720857	B2	20000615		
	EP 866718	A1	19980930	EP 1996-946131	19961213
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2000503004	T2	20000314	JP 1997-523835	19961213
PRAI	US 1995-8518P	P	19951213		
	US 1996-19275P	P	19960607		
	WO 1996-US20463	W	19961213		

AB A method and compns. for delivering a compd. to the cytoplasm of a cell are disclosed. The compd. to be delivered may be an antigenic compd., may be linked to a polycationic affinity handle, or both. In one of the

methods disclosed, the B moiety (for cytoplasmic delivery of the A moiety) of a toxin, such as the **anthrax** PA (**protective antigen**) polypeptide, is also provided to enhance delivery of the compd. to the cytoplasm of the cell.

L11 ANSWER 31 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 20
AN 1997:158460 BIOSIS
DN PREV199799457663
TI Crystal structure of the **anthrax** toxin **protective antigen**.
AU Petosa, Carlo (1); **Collier, R. John**; Klimpel, Kurt R.; Leppla, Stephen H.; Liddington, Robert C.
CS (1) Biochemistry Dep., Univ. Leicester, Leicester LE1 7RH UK
SO Nature (London), (1997) Vol. 385, No. 6619, pp. 833-838. ISSN: 0028-0836.
DT Article
LA English
AB **Protective antigen** (PA) is the central component of the three-part protein toxin secreted by Bacillus **anthracis**, the organism responsible for **anthrax**. After proteolytic activation on the host cell surface, PA forms a membrane-inserting heptamer that translocates the toxic enzymes, oedema factor and lethal factor, into the cytosol. PA, which has a relative molecular mass of 83,000 (M-r 83K), can also translocate heterologous proteins, and is being evaluated for use as a general protein delivery system. Here we report the crystal structure of monomeric PA at 2.1 ANG resolution and the water-soluble heptamer at 4.5 ANG resolution. The monomer is organized mainly into antiparallel beta-sheets and has four domains: an amino-terminal domain (domain 1) containing two calcium ions and the cleavage site for activating proteases; a heptamerization domain (domain 2) containing a large flexible loop implicated in membrane insertion; a small domain of unknown function (domain 3); and a carboxy-terminal receptor-binding domain (domain 4). Removal of a 20K amino-terminal fragment from domain 1 allows the assembly of the heptamer, a ring-shaped structure with a negatively charged lumen, and exposes a large hydrophobic surface for binding the toxic enzymes. We propose a model of pH-dependent membrane insertion involving the formation of a porin-like, membrane-spanning beta-barrel.

L11 ANSWER 32 OF 44 CAPLUS COPYRIGHT 2002 ACS
AN 1997:519026 CAPLUS
DN 127:132013
TI **Anthrax** lethal toxin (Bacillus **anthracis**)
AU Hanna, Philip C.; **Collier, R. John**
CS Department Microbiology, Duke University Medical Center, Durham, NC, 27710, USA
SO Guidebook to Protein Toxins and Their Use in Cell Biology (1997), 91-93. Editor(s): Rappuoli, Rino; Montecucco, Cesare. Publisher: Oxford University Press, Oxford, UK. CODEN: 64UWAW
DT Conference; General Review
LA English
AB A review and discussion with 22 refs. **Anthrax** lethal toxin (LeTx) causes the shock-like symptoms obsd. in systemic **anthrax** infections by inducing macrophages to over-express proinflammatory cytokines. LeTx is comprised of two proteins, both of which are required for toxicity. The **protective antigen** (PA) binds to cellular receptors and is responsible for translocation of the lethal factor (LF), the catalytic moiety, across the plasma membrane into the cytosol. Sequence anal. suggest that LF may be a metalloprotease whose substrate remains unidentified.

L11 ANSWER 33 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1997:281831 BIOSIS
DN PREV199799581034
TI **Anthrax** toxin-mediated delivery of Listeria specific CTL epitopes in vivo.
AU Ballard, Jimmy D.; **Collier, R. John**; Starnbach, Michael N.
CS Harvard Med. Sch., Boston, MA USA
SO Abstracts of the General Meeting of the American Society for Microbiology, (1997) Vol. 97, No. 0, pp. 45.
Meeting Info.: 97th General Meeting of the American Society for Microbiology Miami Beach, Florida, USA May 4-8, 1997
ISSN: 1060-2011.
DT Conference; Abstract; Conference
LA English

L11 ANSWER 34 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 21
AN 1996:544875 BIOSIS
DN PREV199699267231
TI **Anthrax** toxin-mediated delivery of a cytotoxic T-cell epitope in vivo.
AU Ballard, Jimmy D. (1); **Collier, R. John**; Starnbach, Michael N.
CS (1) Dep. Microbiol. Mol. Genet., Harvard Medical Sch., 200 Longwood Ave., Boston, MA 02115 USA
SO Proceedings of the National Academy of Sciences of the United States of America, (1996) Vol. 93, No. 22, pp. 12531-12534.
ISSN: 0027-8424.
DT Article
LA English
AB The **protective antigen** (PA) component of **anthrax** toxin mediates entry of the toxin's lethal factor (LF) and edema factor into the cytosolic compartment of mammalian cells. The amino-terminal domain of LF (LFn; 255 amino acids) binds LF to PA, and when fused to heterologous proteins, the LFn domain delivers such proteins to the cytoplasm in the presence of PA. In the current study, we fused a 9-amino acid cytotoxic T-lymphocyte (CTL) epitope (LLO-91-99) from an intracellular pathogen, Listeria monocytogenes, to LFn and measured the ability of the resulting LFn-LLO-91-99 fusion protein to stimulate a CTL response against the epitope in BALB/c mice. As little as 300 fmol of fusion could stimulate a response. The stimulation was PA-dependent and occurred with the peptide fused to either the amino terminus or the carboxyl terminus of LFn. Upon challenge with L. monocytogenes, mice previously injected with LFn-LLO-91-99 and PA showed a reduction of colony-forming units in spleen and liver, relative to nonimmunized control mice. These results indicate that **anthrax** toxin may be useful as a CTL-peptide delivery system for research and medical applications.

L11 ANSWER 35 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 22
AN 1996:418121 BIOSIS
DN PREV199699140477
TI Fused polycationic peptide mediates delivery of diphtheria toxin A chain to the cytosol in the presence of **anthrax protective antigen**.
AU Blanke, Steven R.; Milne, Jill C.; Benson, Ericka L.; **Collier, R. John (1)**
CS (1) Dep. Microbiol., Mol. Genetics, Harvard Med. Sch., 200 Longwood Ave., Boston, MA 02115 USA
SO Proceedings of the National Academy of Sciences of the United States of America, (1996) Vol. 93, No. 16, pp. 8437-8442.
ISSN: 0027-8424.
DT Article

LA English
AB The lethal factor (LF) and edema factor (EF) of **anthrax** toxin bind by means of their amino-terminal domains to **protective antigen** (PA) on the surface of toxin-sensitive cells and are translocated to the cytosol, where they act on intracellular targets. Genetically fusing the aminoterminal domain of LF LF-N; residues 1-255) to certain heterologous proteins has been shown to potentiate these proteins for PA-dependent delivery to the cytosol. We report here that short tracts of IN-sine, arginine, or histidine residues can also potentiate a protein for such PA-dependent delivery, Fusion of these polycationic tracts to the amino terminus of the enzymic A chain of diphtheria toxin (DTA; residues 1-193) enabled it to be translocated to the cytosol by PA and inhibit protein synthesis. The efficiency of translocation was dependent on tract length: (LF-N gt Lys-8 gt Lys-6 gt Lys-3). Lys-6 was apprxeq 100-fold more active than Arg-6 or His-6, whereas Glu-6 and (SerSerGly)-2 were inactive. Arg-6DTA was partially degraded in cell culture, which may explain its low activity relative to chat of Lys-6DTA. The polycationic tracts may bind to anionic sites at the cell surface (possibly on PA), allowing the fusion proteins to be coendocytosed with PA and delivered to the endosome, where translocation to the cytosol occurs. Excess free LF-N blocked the action of LF-NDTA, but not of Lys-6DTA. This implies that binding to the LF/EF site is not an obligatory step in translocation and suggests that the polycationic tag binds to a different site. Besides elucidating the process of translocation in **anthrax** toxin, these findings may aid in developing systems to deliver heterologous proteins and peptides to the cytoplasm of mammalian cells.

L11 ANSWER 36 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 23
AN 1995:439335 BIOSIS
DN PREV199598453635
TI Effect of **anthrax** toxin's factor on ion channels formed by the **protective antigen.**
AU Zhao, Jianmin; Milne, Jill C.; **Collier, R. John (1)**
CS (1) Shipley Inst. Med., Harvard Med. Sch., Boston, MA 02115 USA
SO Journal of Biological Chemistry, (1995) Vol. 270, No. 31, pp. 18626-18630. ISSN: 0021-9258.
DT Article
LA English
AB **Protective antigen** (PA), a component of **anthrax** toxin, mediates translocation of the toxin's lethal and edema factors (LF and EF, respectively) to the cytoplasm, via a pathway involving their release from an acidic intracellular compartment. PA-63, a 63-kDa proteolytic fragment of PA, can be induced to form ion-conductive channels in the plasma membrane of mammalian cells by acidification of the medium. These channels are believed to be comprised of dodecyl sulfate-resistant oligomers (heptameric rings) of PA-63 seen by electron microscopy of the purified protein. Here we report that the PA-63-mediated efflux of 86Rb+ from preloaded CHO-K1 cells under acidic conditions is strongly inhibited (gtoreq 70%) by LF or LF-N, a PA-binding fragment of LF. Control proteins caused no inhibition. Evidence is presented that the inhibition involves partial blockage of ion conductance by the PA-63 channel. Also, oligomer formation is slowed somewhat by LF at pH values near the pH threshold of channel formation (pH apprx 5.3), suggesting that channel formation may also be retarded under these conditions. The relevance of these results to the location of the LF-binding site on PA-63 and the mechanism of LF and EF translocation is discussed.

L11 ANSWER 37 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1995:286307 BIOSIS
DN PREV199598300607
TI **Anthrax** toxin lethal factor inhibits ion channel activity of

protective antigen in the plasma membrane of CHO-K1 cells.
AU Zhao, Jianmin; Milne, Jill C.; **Collier, R. John**
SO FASEB Journal, (1995) Vol. 9, No. 6, pp. A1314.
Meeting Info.: Annual Meeting of the American Society for Biochemistry and Molecular Biology San Francisco, California, USA May 21-25, 1995
ISSN: 0892-6638.
DT Conference
LA English

L11 ANSWER 38 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 24
AN 1995:203456 BIOSIS
DN PREV199598217756
TI **Protective antigen**-binding domain of **anthrax** lethal factor mediates translocation of a heterologous protein fused to its amino- or carboxy-terminus.
AU Milne, Jill C.; Blanke, Steven R.; Hanna, Philip C.; **Collier, R. John (1)**
CS (1) Dep. Microbiol. Mol. Genetics, Shipley Inst. Med., Harvard Med. Sch., Boston, MA 02115 USA
SO Molecular Microbiology, (1995) Vol. 15, No. 4, pp. 661-666.
ISSN: 0950-382X.
DT Article
LA English
AB The edema factor (EF) and lethal factor (LF) components of **anthrax** toxin require **anthrax protective antigen** (PA) for binding and entry into mammalian cells. After internalization by receptor-mediated endocytosis, PA facilitates the translocation of EF and LF across the membrane of an acidic intracellular compartment. To characterize the translocation process, we generated chimeric proteins composed of the PA recognition domain of LF (LF-N; residues 1-255) fused to either the amino-terminus or the carboxy-terminus of the catalytic chain of diphtheria toxin (DTA). The purified fusion proteins retained ADP-ribosyltransferase activity and reacted with antisera against LF and diphtheria toxin. Both fusion proteins strongly inhibited protein synthesis in CHO-K1 cells in the presence of PA, but not in its absence, and they showed similar levels of activity. This activity could be inhibited by adding LF or the LF-N fragment (which blocked the interaction of the fusion proteins with PA), by adding inhibitors of endosome acidification known to block entry of EF and LF into cells, or by introducing mutations that attenuated the ADP-ribosylation activity of the DTA moiety. The results demonstrate that LF-N fused to either the amino-terminus or the carboxy-terminus of a heterologous protein retains its ability to complement PA in mediating translocation of the protein to the cytoplasm. Besides its importance in understanding translocation, this finding provides the basis for constructing a translocation vector that mediates entry of a variety of heterologous proteins, which may require a free amino- or carboxy-terminus for biological activity, Into the cytoplasm of mammalian cells.

L11 ANSWER 39 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1995:238814 BIOSIS
DN PREV199598253114
TI Membrane translocation by **anthrax** toxins.
AU Milne, Jill C.; Zhao, Jianmin; Ballard, Jimmy; **Collier, R. John**
CS Dep. Microbiol. and Molecular Genetics, Harv. Med. Sch., Boston, MA 02115 USA
SO Abstracts of Papers American Chemical Society, (1995) Vol. 209, No. 1-2, pp. AGFD 13.
Meeting Info.: 209th American Chemical Society National Meeting Anaheim, California, USA April 2-6, 1995

or equal to 70%) by LF or LF sub(N), a PA-binding fragment of LF. Control proteins caused no inhibition. Evidence is presented that the inhibition involves partial blockage of ion conductance by the PA sub(63) channel. Also, oligomer formation is slowed somewhat by LF at pH values near the pH threshold of channel formation (pH similar to 5.3), suggesting that channel formation may also be retarded under these conditions. The relevance of these results to the location of the LF-binding site on PA sub(63) and the mechanism of LF and EF translocation is discussed.

L11 ANSWER 42 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 26
AN 1994:33866 BIOSIS
DN PREV199497046866
TI PH-dependent permeabilization of the plasma membrane of mammalian cells by **anthrax protective antigen**.
AU Milne, Jill C.; **Collier, R. John (1)**
CS (1) Shipley Inst. Med., Harvard Med. Sch., 200 Longwood Ave., Boston, MA 02115 USA
SO Molecular Microbiology, (1993) Vol. 10, No. 3, pp. 647-653.
ISSN: 0950-382X.
DT Article
LA English
AB **Protective antigen** (PA) of **anthrax** toxin forms ion-conductive channels in planar lipid bilayers and liposomes under acidic pH conditions. We show here that PA has a similar permeabilizing action on the plasma membranes of CHO-K1 and three other mammalian cell lines (J774A.1, RAW264.7 and Vero). Changes in membrane permeability were evaluated by measuring the efflux of the K+ analogue, 86Rb+, from prelabelled cells, and the influx of 22Na+. The permeabilizing activity of PA was limited to a proteolytically activated form (PA-N) and was dependent on acidic pH for membrane insertion (optimal at pH 5.0), but not for sustained ion flux. The flux was reduced in the presence of several known channel blockers: tetrabutyl-, tetrapentyl-, and tetrahexylammonium bromides. PA-N facilitated the membrane translocation of **anthrax** edema factor under the same conditions that induced changes in membrane permeability to ions. These results indicate that PA-N permeabilizes cellular membranes under conditions that are believed to prevail in the endosomal compartment of toxin-sensitive cells; and they provide a basis for more detailed studies of the relationship between channel formation and translocation of toxin effector moieties in vivo.

L11 ANSWER 43 OF 44 CAPLUS COPYRIGHT 2002 ACS
AN 1992:2018 CAPLUS
DN 116:2018
TI **Anthrax protective antigen** interacts with a specific receptor on the surface of CHO-K1 cells
AU Escuyer, Vincent; **Collier, R. John**
CS Dep. Microbiol. Mol. Genet., Harvard Med. Sch., Boston, MA, 02115, USA
SO Infect. Immun. (1991), 59(10), 3381-6
CODEN: INFIBR; ISSN: 0019-9567
DT Journal
LA English
AB The interaction of **protective antigen** (PA), a component of the **anthrax** toxin, with receptors of the Chinese hamster ovary cell line CHO-K1 was characterized. **Protective antigen** binding at 4.degree. is highly specific, concn.-dependent, saturable (Kd = 0.9 nM), and reversible. Scatchard anal. indicates the presence of a single class of PA binding sites at a concn. of 10,000 per cell. Pretreatment of cells with a no. of different proteases strongly inhibits PA binding, suggesting that the receptor may be at least partially proteinaceous. Direct chem. crosslinking of radioiodinated PA to the cell surface results in the appearance of a major band exhibiting

an apparent mol. mass of 170 kDa, as estd. by SDS-PAGE. The appearance of this band is completely inhibited by a 200-fold molar excess of unlabeled PA, indicating a high specificity for this interaction. The results suggest that a cell surface protein(s) of 85 to 90 kDa is, or constitutes a portion of, a specific receptor for the PA.

L11 ANSWER 44 OF 44 CAPLUS COPYRIGHT 2002 ACS
AN 1989:207546 CAPLUS
DN 110:207546
TI **Anthrax** toxin: channel-forming activity of **protective antigen** in planar phospholipid bilayers
AU Blaustein, Robert O.; Koehler, Theresa M.; **Collier, R. John**; Finkelstein, Alan
CS Dep. Physiol., Albert Einstein Coll. Med., Bronx, NY, 10461, USA
SO Proc. Natl. Acad. Sci. U. S. A. (1989), 86(7), 2209-13
CODEN: PNASA6; ISSN: 0027-8424
DT Journal
LA English
AB The three sep. proteins that make up **anthrax** toxin - **protective antigen** (PA), edema factor (EF), and lethal factor (LF) - act in binary combinations to produce two distinct reactions in exptl. animals: edema (PA + EF) and death (PA + LF). PA is believed to interact with a membrane receptor, and after proteolytic processing, to mediate endocytosis and subsequent translocation of EF or LF into the cytosol. PA can be sepd., after mild trypsinolysis, into two fragments, PA65 (65 kDa) and PA20 (20 kDa). Trypsin-cleaved PA is capable of forming cation-selective channels in planar phospholipid bilayer membranes; this activity is confined to the PA65 fragment; PA20, LF, and EF are devoid of channel-forming activity. These PA65 channels exhibit pH-dependent and voltage-dependent activity-a property reminiscent of the channels formed by the two-chain proteins diphtheria, tetanus, and botulinum toxins.

=> d his

(FILE 'HOME' ENTERED AT 16:14:58 ON 29 AUG 2002)

FILE 'BIOSIS, MEDLINE, AGRICOLA, EMBASE, CABA, WPIDS, JAPIO, BIOTECHDS, LIFESCI, CAPLUS, USPATFULL, USPAT2' ENTERED AT 16:15:12 ON 29 AUG 2002

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                E COLLIER R JOHN/AU
L1          293 S E1-E3
                E BRADLEY KENNETH A/AU
L2           11 S E2-E3
                E BRADLEY K A/AU
L3          257 S E2-E3
                E MOGRIDGE JEREMY/AU
L4           30 S E3
                E MORGRIDGE J/AU
                E MOGRIDGE J/AU
L5           50 S E3
                E YOUNG JOHNA T/AU
                E YOUNG JOHN A T/AU
L6           76 S E3
                E YOUNGJ A T/AU
                E YOUNG J A T/AU
L7           99 S E3-E4
L8          785 S L1-L7
L9          117 S L8 AND ANTHRA?
L10          92 S L9 AND PROTECTIVE ANTIGEN
L11          44 DUP REM L10 (48 DUPLICATES REMOVED)
```

=> s l11 and receptor

7 FILES SEARCHED...
L12 13 L11 AND RECEPTOR

=> d bib 1-13

L12 ANSWER 1 OF 13 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2002:364347 BIOSIS
DN PREV200200364347
TI 2001: A year of major advances in **anthrax** toxin research.
AU Mourez, Michael (1); Lacy, D. Borden (1); Cunningham, Kristina (1); Legmann, Rachel (1); Sellman, Bret R.; **Mogridge, Jeremy; Collier, R. John (1)**
CS (1) Dept of Microbiology and Molecular Genetics, Harvard Medical School, 200 Longwood Ave, Boston, MA, 02115: jcollier@hms.harvard.edu USA
SO Trends in Microbiology, (June, 2002) Vol. 10, No. 6, pp. 287-293. http://journals.bmn.com/journals/list/latest?jcode=tim. print. ISSN: 0966-842X.
DT General Review
LA English

L12 ANSWER 2 OF 13 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2002:340526 BIOSIS
DN PREV200200340526
TI The lethal and edema factors of **anthrax** toxin bind only to oligomeric forms of the **protective antigen**.
AU **Mogridge, Jeremy**; Cunningham, Kristina; Lacy, D. Borden; Mourez, Michael; **Collier, R. John (1)**
CS (1) Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA, 02115: jcollier@hms.harvard.edu USA
SO Proceedings of the National Academy of Sciences of the United States of America, (May 14, 2002) Vol. 99, No. 10, pp. 7045-7048. http://www.pnas.org. print. ISSN: 0027-8424.
DT Article
LA English

L12 ANSWER 3 OF 13 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2001:566770 BIOSIS
DN PREV200100566770
TI Identification of the cellular **receptor** for **anthrax** toxin.
AU **Bradley, Kenneth A.; Mogridge, Jeremy**; Mourez, Michael; **Collier, R. John; Young, John A. T. (1)**
CS (1) McArdle Laboratory for Cancer Research, University of Wisconsin-Madison, 1400 University Avenue, Madison, WI, 53706: young@oncology.wisc.edu USA
SO Nature (London), (8 November, 2001) Vol. 414, No. 6860, pp. 225-229. print. ISSN: 0028-0836.
DT Article
LA English
SL English

L12 ANSWER 4 OF 13 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2001:238485 BIOSIS
DN PREV200100238485
TI Point mutations in **anthrax protective antigen** that block translocation.
AU Sellman, Bret R.; Nassi, Shilla; **Collier, R. John (1)**
CS (1) Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA, 02115: jcollier@hms.harvard.edu USA
SO Journal of Biological Chemistry, (March 16, 2001) Vol. 276, No. 11, pp.

AN 1992:2018 CAPLUS
DN 116:2018
TI **Anthrax protective antigen** interacts with a specific **receptor** on the surface of CHO-K1 cells
AU Escuyer, Vincent; **Collier, R. John**
CS Dep. Microbiol. Mol. Genet., Harvard Med. Sch., Boston, MA, 02115, USA
SO Infect. Immun. (1991), 59(10), 3381-6
CODEN: INFIBR; ISSN: 0019-9567
DT Journal
LA English

L12 ANSWER 13 OF 13 CAPLUS COPYRIGHT 2002 ACS
AN 1989:207546 CAPLUS
DN 110:207546
TI **Anthrax** toxin: channel-forming activity of **protective antigen** in planar phospholipid bilayers
AU Blaustein, Robert O.; Koehler, Theresa M.; **Collier, R. John**; Finkelstein, Alan
CS Dep. Physiol., Albert Einstein Coll. Med., Bronx, NY, 10461, USA
SO Proc. Natl. Acad. Sci. U. S. A. (1989), 86(7), 2209-13
CODEN: PNASA6; ISSN: 0027-8424
DT Journal
LA English

=> s anthra?
L13 268052 ANTHRA?

=> s l13 and protective (5a) antigen
L14 1553 L13 AND PROTECTIVE (5A) ANTIGEN

=> s l14 and receptor
L15 314 L14 AND RECEPTOR

=> dup rem l15
PROCESSING COMPLETED FOR L15
L16 123 DUP REM L15 (191 DUPLICATES REMOVED)

=> d bib 1-123

L16 ANSWER 1 OF 123 CAPLUS COPYRIGHT 2002 ACS
AN 2002:449716 CAPLUS
DN 137:29035
TI Sequences of a human **receptor** for B. **anthracis** toxin and therapeutical uses
IN Young, John A. T.; Bradley, Kenneth A.; Collier, Robert J.; Mogridge, Jeremy S.
PA Wisconsin Alumni Research Foundation, USA
SO PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002046228	A2	20020613	WO 2001-US30941	20011003

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
PRAI US 2000-251481P P 20001205

L16 ANSWER 2 OF 123 USPATFULL
AN 2002:172486 USPATFULL
TI Dendritic cell co-stimulatory molecules
IN Pardoll, Drew M., Brookville, MD, UNITED STATES
Tsuchiya, Haruo, Baltimore, MD, UNITED STATES
Gorski, Kevin S., Baltimore, MD, UNITED STATES
Tseng, Su-Yi, Baltimore, MD, UNITED STATES
PI US 2002091246 A1 20020711
AI US 2001-794210 A1 20010228 (9)
PRAI US 2000-200580P 20000428 (60)
US 2000-240169P 20001013 (60)
DT Utility
FS APPLICATION
LREP VENABLE, Post Office Box 34385, Washington, DC, 20043-9998
CLMN Number of Claims: 120
ECL Exemplary Claim: 1
DRWN 8 Drawing Page(s)
LN.CNT 3534
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 3 OF 123 USPATFULL
AN 2002:105667 USPATFULL
TI Inhibition of mitogen-activated protein kinase (MAPK) pathway: a selective therapeutic strategy against melanoma
IN Koo, Han-Mo, Kentwood, MI, UNITED STATES
Vande Woude, George F., Ada, MI, UNITED STATES
PI US 2002054869 A1 20020509
AI US 2001-942940 A1 20010831 (9)
PRAI US 2000-229290P 20000901 (60)
US 2001-285690P 20010424 (60)
DT Utility
FS APPLICATION
LREP VENABLE, BAETJER, HOWARD AND CIVILETTI, LLP, P.O. BOX 34385, WASHINGTON, DC, 20043-9998
CLMN Number of Claims: 21
ECL Exemplary Claim: 1
DRWN 16 Drawing Page(s)
LN.CNT 2335
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 4 OF 123 USPATFULL
AN 2002:98896 USPATFULL
TI Methods for protection against lethal infection with bacillus **anthracis**
IN Galloway, Darrel R., Dublin, OH, UNITED STATES
Mateczun, Alfred J., Albuquerque, NM, UNITED STATES
PI US 2002051791 A1 20020502
AI US 2000-747521 A1 20001221 (9)
PRAI US 1999-171459P 19991222 (60)
DT Utility
FS APPLICATION
LREP NAVAL MEDICAL RESEARCH CENTER, ATTN: (CODE 00L), 503 ROBERT GRANT AVENUE, SILVER SPRING, MD, 20910-7500
CLMN Number of Claims: 30
ECL Exemplary Claim: 1
DRWN 8 Drawing Page(s)
LN.CNT 1459

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 5 OF 123 USPATFULL
AN 2002:92073 USPATFULL
TI Targeting antigens to the MHC class I processing pathway with an **anthrax** toxin fusion protein
IN Klimpel, Kurt, Gaithersburg, MD, UNITED STATES
Goletz, Theresa J., Kensington, MD, UNITED STATES
Arora, Naveen, Delhi, INDIA
Leppla, Stephen H., Bethesda, MD, UNITED STATES
Berzofsky, Jay A., Bethesda, MD, UNITED STATES
PI US 2002048590 A1 20020425
AI US 2001-853530 A1 20010509 (9)
RLI Division of Ser. No. US 1997-937276, filed on 15 Sep 1997, PENDING
PRAI US 1996-25270P 19960917 (60)
DT Utility
FS APPLICATION
LREP TOWNSEND AND TOWNSEND AND CREW, TWO EMBARCADERO CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834
CLMN Number of Claims: 28
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1192
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 6 OF 123 USPATFULL
AN 2002:72451 USPATFULL
TI Compounds and methods for the treatment and prevention of bacterial infection
IN Collier, R. John, Wellesley, MA, UNITED STATES
Sellman, Bret R., Rochester, NY, UNITED STATES
PI US 2002039588 A1 20020404
AI US 2001-848909 A1 20010504 (9)
PRAI US 2000-201800P 20000504 (60)
DT Utility
FS APPLICATION
LREP CLARK & ELBING LLP, 176 FEDERAL STREET, BOSTON, MA, 02110-2214
CLMN Number of Claims: 28
ECL Exemplary Claim: 1
DRWN 22 Drawing Page(s)
LN.CNT 1502
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 7 OF 123 USPATFULL
AN 2002:48266 USPATFULL
TI Single target counting assays using semiconductor nanocrystals
IN Empedocles, Stephen Alexander, Mountain View, CA, UNITED STATES
Watson, Andrew R., Belmont, CA, UNITED STATES
Phillips, Vince, Sunnyvale, CA, UNITED STATES
Wong, Edith, Danville, CA, UNITED STATES
PA Quantum Dot Corporation, Hayward, CA, UNITED STATES, 94545 (U.S. corporation)
PI US 2002028457 A1 20020307
AI US 2001-882193 A1 20010613 (9)
RLI Continuation-in-part of Ser. No. US 2001-784866, filed on 15 Feb 2001, PENDING
PRAI US 2000-182844P 20000216 (60)
US 2000-211054P 20000613 (60)
DT Utility
FS APPLICATION
LREP TOWNSEND AND TOWNSEND AND CREW, LLP, TWO EMBARCADERO CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834

CLMN Number of Claims: 18
ECL Exemplary Claim: 1
DRWN 15 Drawing Page(s)
LN.CNT 2844
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 8 OF 123 USPATFULL
AN 2002:37316 USPATFULL
TI Immuno-adjuvant PDT treatment of metastatic tumors
IN Curry, Patrick Mark, Vancouver, CANADA
Richter, Anna M., Vancouver, CANADA
Levy, Julia G., Vancouver, CANADA
Hunt, David W.C., White Rock, CANADA
PI US 2002022032 A1 20020221
AI US 2001-756687 A1 20010109 (9)
RLI Continuation-in-part of Ser. No. US 2000-556833, filed on 21 Apr 2000, PENDING
PRAI US 1999-130519P 19990423 (60)
DT Utility
FS APPLICATION
LREP MORRISON & FOERSTER LLP, 3811 VALLEY CENTRE DRIVE, SUITE 500, SAN DIEGO, CA, 92130-2332
CLMN Number of Claims: 27
ECL Exemplary Claim: 1
DRWN 4 Drawing Page(s)
LN.CNT 2765
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 9 OF 123 USPATFULL
AN 2002:209522 USPATFULL
TI Inhibitors of **anthrax** lethal factor activity
IN Rideout, Darryl, San Diego, CA, United States
Yalamoori, Venkatachalapathi V., San Diego, CA, United States
Ramnarayan, Kalyanaraman, San Diego, CA, United States
Shenderovich, Mark, San Diego, CA, United States
Zheng, Jian Hua, San Diego, CA, United States
Sun, Jason, San Diego, CA, United States
Niemeyer, Christina, San Diego, CA, United States
PA Structural Bioinformatics Inc., San Diego, CA, United States (U.S. corporation)
PI US 6436933 B1 20020820
AI US 2001-818259 20010326 (9)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Rose, Shep K.; Assistant Examiner: Jagoe, Donna
LREP Weseman, Esq., James C., The Law Offies of James C. Weseman
CLMN Number of Claims: 3
ECL Exemplary Claim: 1
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 1426

L16 ANSWER 10 OF 123 USPATFULL
AN 2002:201863 USPATFULL
TI Dendritic cell **receptor**
IN Hart, Derek N., Christchurch, NEW ZEALAND
PA The Corporation of the Trustees of the Sisters of Mercy in Queensland, Queensland, AUSTRALIA (non-U.S. corporation)
PI US 6432666 B1 20020813
WO 9745449 19971204
AI US 1999-194612 19990318 (9)
WO 1997-NZ68 19970529
19990318 PCT 371 date

PRAI NZ 1996-286692 19960529
DT Utility
FS GRANTED
EXNAM Primary Examiner: Eyler, Yvonne; Assistant Examiner: Hamud, Fozia
LREP Nixon & Vanderhye
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN 19 Drawing Figure(s); 15 Drawing Page(s)
LN.CNT 1781

L16 ANSWER 11 OF 123 USPATFULL
AN 2002:188260 USPATFULL
TI Analyte sensing mediated by adapter/carrier molecules
IN Bayley, Hagan, College Station, TX, United States
Braha, Orit, College Station, TX, United States
Gu, LiQun, Bryan, TX, United States
PA The Texas A&M University System, College Station, TX, United States (U.S. corporation)
PI US 6426231 B1 20020730
AI US 1999-441376 19991117 (9)
PRAI US 1998-109034P 19981118 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Chin, Christopher L.
LREP Baker Botts L.L.P.
CLMN Number of Claims: 39
ECL Exemplary Claim: 1
DRWN 33 Drawing Figure(s); 9 Drawing Page(s)
LN.CNT 1747
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 12 OF 123 USPATFULL
AN 2002:136555 USPATFULL
TI Methods of modulating an immune response to antigen, and cells for use in the method
IN Segal, Andrew H., Boston, MA, United States
PA Whitehead Institute for Biomedical Research, Cambridge, MA, United States (U.S. corporation)
PI US 6403080 B1 20020611
AI US 1999-339523 19990624 (9)
RLI Division of Ser. No. US 1997-826259, filed on 27 Mar 1997, now patented, Pat. No. US 5951976
PRAI US 1996-14364P 19960328 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Bansal, Geetha P.
LREP Williams, Kathleen Madden, Palmer & Dodge, LLP
CLMN Number of Claims: 25
ECL Exemplary Claim: 1
DRWN 0 Drawing Figure(s); 0 Drawing Page(s)
LN.CNT 2153
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 13 OF 123 USPATFULL
AN 2002:50802 USPATFULL
TI Computer readable genomic sequence of Haemophilus influenzae Rd, fragments thereof, and uses thereof
IN Fleischmann, Robert D., Gaithersburg, MD, United States
Adams, Mark D., N. Potomac, MD, United States
White, Owen, Gaithersburg, MD, United States
Smith, Hamilton O., Towson, MD, United States
Venter, J. Craig, Potomac, MD, United States

PA Human Genome Sciences, Inc., Rockville, MD, United States (U.S. corporation)
PI US 6355450 B1 20020312
AI US 1995-476102 19950607 (8)
RLI Continuation-in-part of Ser. No. US 1995-426787, filed on 21 Apr 1995, now abandoned
DT Utility
FS GRANTED
EXNAM Primary Examiner: Campell, Bruce R.
CLMN Number of Claims: 88
ECL Exemplary Claim: 1
DRWN 47 Drawing Figure(s); 47 Drawing Page(s)
LN.CNT 4666
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 14 OF 123 USPATFULL
AN 2002:34423 USPATFULL
TI Noninvasive genetic immunization, expression products therefrom and uses thereof
IN Tang, De-chu C., Birmingham, AL, United States
Marks, Donald H., Rockaway, NJ, United States
Curiel, David T., Birmingham, AL, United States
Shi, Zhongkai, Birmingham, AL, United States
van Kampen, Kent Rigby, Hoover, AL, United States
PA The UAB Research Foundation, Birmingham, AL, United States (U.S. corporation)
PI US 6348450 B1 20020219
AI US 2000-563826 20000503 (9)
RLI Continuation-in-part of Ser. No. US 2000-533149, filed on 23 Mar 2000 Continuation-in-part of Ser. No. US 402527 Continuation-in-part of Ser. No. WO 1998-US16739, filed on 13 Aug 1998
PRAI US 1999-132216P 19990503 (60)
US 1998-75113P 19980211 (60)
US 1997-55520P 19970813 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Crouch, Deborah; Assistant Examiner: Woitach, Joseph T.
LREP Frommer Lawrence & Haug LLP, Frommer, William S., Kowalski, Thomas J.
CLMN Number of Claims: 52
ECL Exemplary Claim: 1
DRWN 21 Drawing Figure(s); 19 Drawing Page(s)
LN.CNT 2393
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 15 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 1
AN 2002:340526 BIOSIS
DN PREV200200340526
TI The lethal and edema factors of **anthrax** toxin bind only to oligomeric forms of the **protective antigen**.
AU Mogridge, Jeremy; Cunningham, Kristina; Lacy, D. Borden; Mourez, Michael; Collier, R. John (1)
CS (1) Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA, 02115: jcollier@hms.harvard.edu USA
SO Proceedings of the National Academy of Sciences of the United States of America, (May 14, 2002) Vol. 99, No. 10, pp. 7045-7048.
http://www.pnas.org. print.
ISSN: 0027-8424.
DT Article
LA English

AN 2002-06073 BIOTECHDS
TI Screening Bacillus **anthracis** toxicity inhibitor (T) by generating recombinant **protective antigen** 32, comparing fluorescence of cells contacted with PA32-fluorescent marker fusion protein before, after contact with T; vector-mediated **protective antigen**-32 and enhanced green fluorescent protein reporter gene transfer, expression in human A549 cell, single chain antibody and nucleic acid vaccine for recombinant protein production, drugscreening and bacterium infection therapy and gene therapy
AU CIRINO N M; JACKSON P J; LEHNERT B E
PA UNIV CALIFORNIA
PI US 6329156 11 Dec 2001
AI US 1999-273839 22 Mar 1999
PRAI US 1999-273839 22 Mar 1999
DT Patent
LA English
OS WPI: 2002-121130 [16]

L16 ANSWER 21 OF 123 WPIDS (C) 2002 THOMSON DERWENT
AN 2002-017725 [02] WPIDS
DNN N2002-014125 DNC C2002-005170
TI Protecting humans against **anthrax** using mutant B groups (**anthrax** protective antigens) of the pore-forming binary A-B toxin of Bacillus **anthracis**.
DC B04 D16 P31
IN COLLIER, R J; SELLMAN, B R
PA (HARD) HARVARD COLLEGE; (COLL-I) COLLIER R J; (SELL-I) SELLMAN B R
CYC 95
PI WO 2001082788 A2 20011108 (200202)* EN 75p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2001061171 A 20011112 (200222)
US 2002039588 A1 20020404 (200227)
ADT WO 2001082788 A2 WO 2001-US14372 20010504; AU 2001061171 A AU 2001-61171 20010504; US 2002039588 A1 Provisional US 2000-201800P 20000504, US 2001-848909 20010504
FDT AU 2001061171 A Based on WO 200182788
PRAI US 2000-201800P 20000504; US 2001-848909 20010504

L16 ANSWER 22 OF 123 WPIDS (C) 2002 THOMSON DERWENT
AN 2001-218343 [22] WPIDS
DNC C2001-065177
TI Novel fusion protein for modifying apoptosis in target cell and reducing apoptosis after transient ischemic neuronal injury, has two domains which targets protein to a cell and modifies apoptotic response of cell.
DC B04 D16
IN COLLIER, R J; LIU, X; YOULE, R J
PA (HARD) HARVARD COLLEGE; (USSH) US DEPT HEALTH & HUMAN SERVICES
CYC 94
PI WO 2001012661 A2 20010222 (200122)* EN 55p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2000069061 A 20010313 (200134)

ADT WO 2001012661 A2 WO 2000-US22293 20000815; AU 2000069061 A AU 2000-69061 20000815
FDT AU 2000069061 A Based on WO 200112661
PRAI US 1999-149220P 19990816

L16 ANSWER 23 OF 123 BIOTECHDS COPYRIGHT 2002 THOMSON DERWENT AND ISI
AN 2001-08818 BIOTECHDS
TI Targeting compounds typically lethal factor polypeptide to cells for prophylactic by using mutant **protective antigen** proteins that target cells containing high amounts of cell-surface metallo proteases or plasminogen-activators; fusion protein of lethal factor for use in diagnosis and therapy
AU Leppla S H; Liu S H; Netzel-Arnett S; Hansen-Birkedal H; Bugge T
PA U.S.Dep.Health-Hum.Serv.
LO Rockville, MD, USA.
PI WO 2001021656 29 Mar 2001
AI WO 2000-US26192 22 Sep 2000
PRAI US 1999-155961 24 Sep 1999
DT Patent
LA English
OS WPI: 2001-257973 [26]

L16 ANSWER 24 OF 123 BIOTECHDS COPYRIGHT 2002 THOMSON DERWENT AND ISI
AN 2001-07648 BIOTECHDS
TI Novel fusion protein for modifying apoptosis in target cell and reducing apoptosis after transient ischemic neuronal injury, has 2 domains which targets protein to a cell and modifies apoptotic response of cell; plasmid pcDNA3-mediated diphtheria toxin **receptor** binding domain and BCL-xl domain gene transfer and expression in Escherichia coli
AU Youle R J; Liu X; Collier R J
PA U.S.Dep.Health-Hum.Serv.; Nat.Inst.Health-Rockville; Univ.Harvard
LO Rockville, MD, USA; Cambridge, MA, USA.
PI WO 2001012661 22 Feb 2001
AI WO 2000-US22293 15 Aug 2000
PRAI US 1999-149220 16 Aug 1999
DT Patent
LA English
OS WPI: 2001-218343 [22]

L16 ANSWER 25 OF 123 USPATFULL
AN 2001:182107 USPATFULL
TI Vaccine compositions and methods of modulating immune responses
IN Segal, Andrew, Cambridge, MA, United States
PI US 2001031264 A1 20011018
AI US 2001-789922 A1 20010221 (9)
RLI Continuation-in-part of Ser. No. US 1998-7711, filed on 15 Jan 1998, GRANTED, Pat. No. US 6224870
PRAI US 1996-11047P 19960125 (60)
DT Utility
FS APPLICATION
LREP PALMER & DODGE, LLP, ONE BEACON STREET, BOSTON, MA, 02108-3190
CLMN Number of Claims: 7
ECL Exemplary Claim: 1
DRWN 2 Drawing Page(s)
LN.CNT 2512
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 26 OF 123 USPATFULL
AN 2001:170889 USPATFULL
TI Monocyte-derived dendritic cell subsets
IN Punnonen, Juha, Palo Alto, CA, United States

Chang, Chia-Chun J., Los Gatos, CA, United States
PI US 2001026937 A1 20011004
AI US 2001-760388 A1 20010110 (9)
PRAI US 2000-175552P 20000111 (60)
US 2000-181957P 20000210 (60)
DT Utility
FS APPLICATION
LREP LAW OFFICES OF JONATHAN ALAN QUINE, P O BOX 458, ALAMEDA, CA, 94501
CLMN Number of Claims: 69
ECL Exemplary Claim: 1
DRWN 7 Drawing Page(s)
LN.CNT 3189

L16 ANSWER 27 OF 123 USPATFULL
AN 2001:178820 USPATFULL
TI Organic semiconductor recognition complex and system
IN Kiel, Johnathan L., Universal City, TX, United States
Bruno, John G., San Antonio, TX, United States
Parker, Jill E., Floresville, TX, United States
Alls, John L., San Antonio, TX, United States
Batishko, Charles R., Richland, WA, United States
Holwitt, Eric A., San Antonio, TX, United States
PA Conceptual Mind Works, Inc., San Antonio, TX, United States (U.S. corporation)
PI US 6303316 B1 20011016
AI US 2000-608706 20000630 (9)
PRAI US 1999-142301P 19990702 (60)
US 2000-199620P 20000425 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Horlick, Kenneth R.
LREP Blakely, Sokoloff, Taylor & Zafman
CLMN Number of Claims: 62
ECL Exemplary Claim: 1
DRWN 31 Drawing Figure(s); 15 Drawing Page(s)
LN.CNT 3322
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 28 OF 123 USPATFULL
AN 2001:67794 USPATFULL
TI Human respiratory syncytial virus peptides with antifusogenic and antiviral activities
IN Barney, Shawn O'Lin, Cary, NC, United States
Lambert, Dennis Michael, Cary, NC, United States
Petteway, Stephen Robert, Cary, NC, United States
PA Trimeris, Inc., Durham, NC, United States (U.S. corporation)
PI US 6228983 B1 20010508
AI US 1995-485264 19950607 (8)
RLI Division of Ser. No. US 1995-470896, filed on 6 Jun 1995
Continuation-in-part of Ser. No. US 1994-360107, filed on 20 Dec 1994
Continuation-in-part of Ser. No. US 1994-255208, filed on 7 Jun 1994
Continuation-in-part of Ser. No. US 1993-73028, filed on 7 Jun 1993, now patented, Pat. No. US 5464933
DT Utility
FS Granted
EXNAM Primary Examiner: Scheiner, Laurie; Assistant Examiner: Parkin, Jeffrey S.
LREP Pennie & Edmonds LLP
CLMN Number of Claims: 62
ECL Exemplary Claim: 1
DRWN 84 Drawing Figure(s); 83 Drawing Page(s)
LN.CNT 32166

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 29 OF 123 USPATFULL
AN 2001:63248 USPATFULL
TI Vaccine compositions and methods of modulating immune responses
IN Segal, Andrew H., Boston, MA, United States
PA Genitrix, Ltd., Cambridge, MA, United States (U.S. corporation)
PI US 6224870 B1 20010501
AI US 1998-7711 19980115 (9)
RLI Continuation-in-part of Ser. No. US 1997-788143, filed on 24 Jan 1997, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Saunders, David; Assistant Examiner: DeCloux, Amy
LREP Palmer & Dodge, LLP, Williams, Kathleen M.
CLMN Number of Claims: 7
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 2264
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 30 OF 123 USPATFULL
AN 2001:56099 USPATFULL
TI Prostate cancer-specific marker
IN French, Cynthia K., Irvine, CA, United States
Schneider, Patrick A., Irvine, CA, United States
Yamamoto, Karen K., San Clemente, CA, United States
PA Diagnostic Products Corporation, Los Angeles, CA, United States (U.S. corporation)
PI US 6218523 B1 20010417
AI US 1998-36315 19980306 (9)
PRAI US 1997-41246P 19970307 (60)
US 1997-47811P 19970515 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Schwartzman, Robert A.; Assistant Examiner: Schmidt, Mary M.
LREP Mueth, Joseph E.
CLMN Number of Claims: 5
ECL Exemplary Claim: 1
DRWN 8 Drawing Figure(s); 6 Drawing Page(s)
LN.CNT 2368
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 31 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC. DUPLICATE 4
AN 2001:354345 BIOSIS
DN PREV200100354345
TI Targeting of tumor cells by cell surface urokinase plasminogen activator-dependent **anthrax** toxin.
AU Liu, Shihui; Bugge, Thomas H.; Leppla, Stephen H. (1)
CS (1) Oral Infection and Immunity Branch, NIDCR, National Institutes of Health, 30 Convent Dr., MSC 4350, Bldg. 30, Rm. 303, Bethesda, MD, 20892-4350: Leppla@nih.gov USA
SO Journal of Biological Chemistry, (May 25, 2001) Vol. 276, No. 21, pp. 17976-17984. print.
ISSN: 0021-9258.
DT Article
LA English
SL English

L16 ANSWER 32 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

SL English

L16 ANSWER 36 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 9
AN 2001:462977 BIOSIS
DN PREV200100462977
TI Participation of residue F552 in domain III of the **protective antigen** in the biological activity of **anthrax** lethal toxin.
AU Khanna, Hemant; Gupta, Pradeep K.; Singh, Anubha; Chandra, Ramesh; Singh, Yogendra (1)
CS (1) Centre for Biochemical Technology, Mall Road, Delhi, 110007 India
SO Biological Chemistry, (June, 2001) Vol. 382, No. 6, pp. 941-946. print.
ISSN: 1431-6730.
DT Article
LA English
SL English

L16 ANSWER 37 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2002:230400 BIOSIS
DN PREV200200230400
TI War against **anthrax**.
AU Khanna, Hemant; Singh, Yogendra (1)
CS (1) Centre for Biochemical Technology, Mall Road, Delhi, 110007: ysingh@cbt.res.in India
SO Molecular Medicine (Baltimore), (December, 2001) Vol. 7, No. 12, pp. 795-796. print.
ISSN: 1076-1551.
DT Article
LA English

L16 ANSWER 38 OF 123 MEDLINE
AN 2001262891 MEDLINE
DN 21225892 PubMed ID: 11326092
TI Dominant-negative mutants of a toxin subunit: an approach to therapy of **anthrax**.
CM Comment in: Science. 2001 Apr 27;292(5517):647-8
AU Sellman B R; Mourez M; Collier R J
CS Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA 02115, USA.
NC 5T32AI07410 (NIAID)
R37-AI22021 (NIAID)
SO SCIENCE, (2001 Apr 27) 292 (5517) 695-7.
Journal code: 0404511. ISSN: 0036-8075.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200105
ED Entered STN: 20010521
Last Updated on STN: 20010521
Entered Medline: 20010517

L16 ANSWER 39 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 10
AN 2001:493425 BIOSIS
DN PREV200100493425
TI Hydrophobic residues Phe552, Phe554, Ile562, Leu566, and Ile574 are required for oligomerization of **anthrax protective antigen**.
AU Ahuja, Nidhi; Kumar, Praveen; Bhatnagar, Rakesh (1)
CS (1) Centre for Biotechnology, Jawaharlal Nehru University, New Delhi,

L16 ANSWER 43 OF 123 MEDLINE DUPLICATE 13
AN 2001550552 MEDLINE
DN 21480431 PubMed ID: 11596878
TI **Anthrax** toxin.
AU Bhatnagar R; Batra S
CS Centre for Biotechnology, Jawaharlal Nehru University, New Delhi, India.. rakesh@jnuniv.ernet.in
SO CRITICAL REVIEWS IN MICROBIOLOGY, (2001) 27 (3) 167-200. Ref: 194
Journal code: 8914274. ISSN: 1040-841X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English
FS Priority Journals
EM 200202
ED Entered STN: 20011015
Last Updated on STN: 20020301
Entered Medline: 20020228

L16 ANSWER 44 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2002:176584 BIOSIS
DN PREV200200176584
TI **Anthrax** toxin **protective antigen** oligomer is the only form to enter the cells that is dependent upon clathrin-coated pits.
AU Liu, S. (1); Leppla, S. H. (1)
CS (1) National Institute of Dental and Craniofacial Research, NIH, Bethesda, MD USA
SO Abstracts of the General Meeting of the American Society for Microbiology, (2001) Vol. 101, pp. 110. http://www.asmusa.org/mtgsrc/generalmeeting.htm. print.
Meeting Info.: 101st General Meeting of the American Society for Microbiology Orlando, FL, USA May 20-24, 2001
ISSN: 1060-2011.
DT Conference
LA English

L16 ANSWER 45 OF 123 USPATFULL
AN 2000:15631 USPATFULL
TI Methods and reagents for inhibiting furin endoprotease
IN Thomas, Gary, Tualatin, OR, United States
Anderson, Eric D., Portland, OR, United States
Thomas, Laurel, Tualatin, OR, United States
Hayflick, Joel S., Seattle, WA, United States
PA Oregan Health Sciences University, Portland, OR, United States (U.S. corporation)
PI US 6022855 20000208
WO 9416073 19940721
AI US 1995-481534 19950914 (8)
WO 1994-US247 19940107
19950914 PCT 371 date
19950914 PCT 102(e) date
RLI Continuation-in-part of Ser. No. US 1993-2202, filed on 8 Jan 1993, now patented, Pat. No. US 5604201
DT Utility
FS Granted
EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Lau, Kawai
LREP McDonnell Boehnen Hulbert & Berghoff
CLMN Number of Claims: 18
ECL Exemplary Claim: 1
DRWN 17 Drawing Figure(s); 10 Drawing Page(s)

LN.CNT 1677
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 46 OF 123 USPATFULL
AN 2000:9723 USPATFULL
TI Unique nucleotide and amino acid sequence and uses thereof
IN Summers, Max D., Bryan, TX, United States
Braunagel, Sharon C., Bryan, TX, United States
Hong, Tao, Bryan, TX, United States
PA The Texas A & M University System, College Station, TX, United States (U.S. corporation)
PI US 6017734 20000125
AI US 1997-792832 19970130 (8)
RLI Continuation-in-part of Ser. No. US 1996-678435, filed on 3 Jul 1996, now abandoned
PRAI US 1995-955P 19950707 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Elliott, George C.; Assistant Examiner: Schwartzman, Robert
LREP Arnold, White & Durkee
CLMN Number of Claims: 56
ECL Exemplary Claim: 1
DRWN 47 Drawing Figure(s); 24 Drawing Page(s)
LN.CNT 7846
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 47 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 14
AN 2000:393065 BIOSIS
DN PREV200000393065
TI A quantitative study of the interactions of Bacillus **anthracis** edema factor and lethal factor with activated **protective antigen**.
AU Elliott, Jennifer L.; Mogridge, Jeremy; Collier, R. John (1)
CS (1) Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA, 02115 USA
SO Biochemistry, (June 6, 2000) Vol. 39, No. 22, pp. 6706-6713. print. ISSN: 0006-2960.
DT Article
LA English
SL English

L16 ANSWER 48 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 15
AN 2000:182028 BIOSIS
DN PREV200000182028
TI Role of toxin functional domains in **anthrax** pathogenesis.
AU Brossier, Fabien; Weber-Levy, Martine; Mock, Michele (1); Sirard, Jean-Claude
CS (1) Unite Toxines et Pathogenie Bacteriennes, Institut Pasteur, 28, rue du Dr. Roux, 75724, Paris Cedex, 15 France
SO Infection and Immunity, (April, 2000) Vol. 68, No. 4, pp. 1781-1786. ISSN: 0019-9567.
DT Article
LA English
SL English

L16 ANSWER 49 OF 123 CAPLUS COPYRIGHT 2002 ACS
AN 2000:568810 CAPLUS
DN 133:262509
TI Translocation of Bacillus **anthracis** lethal and edema factors

across endosome membranes
AU Guidi-Rontani, Chantal; Weber-Levy, Martine; Mock, Michele; Cabiaux, Veronique
CS Unite Toxines et Pathogenie Bacteriennes, CNRS URA 1858, Institut Pasteur, Paris, 75015, Fr.
SO Cellular Microbiology (2000), 2(3), 259-264
CODEN: CEMIF5; ISSN: 1462-5814
PB Blackwell Science Ltd.
DT Journal
LA English
RE.CNT 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 50 OF 123 MEDLINE DUPLICATE 16
AN 2001337913 MEDLINE
DN 21129592 PubMed ID: 11207581
TI Proteolytic activation of **receptor**-bound **anthrax** **protective antigen** on macrophages promotes its internalization.
AU Beauregard K E; Collier R J; Swanson J A
CS Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA 02115, USA.
NC AI22021 (NIAID)
AI35950 (NIAID)
SO CELLULAR MICROBIOLOGY, (2000 Jun) 2 (3) 251-8.
Journal code: 100883691. ISSN: 1462-5814.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200106
ED Entered STN: 20010618
Last Updated on STN: 20010618
Entered Medline: 20010614

L16 ANSWER 51 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2000:345564 BIOSIS
DN PREV200000345564
TI On the molecular interaction of **anthrax** lethal toxin components.
AU Khanna, H. (1); Chopra, A. P. (1); Leppla, S. H.; Singh, Y. (1)
CS (1) Centre for Biochemical Technology, New Delhi India
SO Abstracts of the General Meeting of the American Society for Microbiology, (2000) Vol. 100, pp. 79. print.
Meeting Info.: 100th General Meeting of the American Society for Microbiology Los Angeles, California, USA May 21-25, 2000 American Society for Microbiology
. ISSN: 1060-2011.
DT Conference
LA English
SL English

L16 ANSWER 52 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 17
AN 2000:476204 BIOSIS
DN PREV200000476204
TI **Anthrax** toxin-mediated delivery of cholera toxin-A subunit into the cytosol of mammalian cells.
AU Sharma, Manju; Khanna, Hemant; Arora, Naveen; Singh, Yogendra (1)
CS (1) Centre for Biochemical Technology, Mall Road, Near Jubilee Hall, Delhi, 110007 India
SO Biotechnology and Applied Biochemistry, (August, 2000) Vol. 32, No. 1, pp. 69-72. print.

ISSN: 0885-4513.
DT Article
LA English
SL English

L16 ANSWER 53 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2001:183929 BIOSIS
DN PREV200100183929
TI Dissection of domain 4 of Bacillus **anthracis protective antigen**: The cellular **receptor** and neutralizing monoclonal antibodies recognize overlapping but distinct regions.
AU Varughese, Mini (1); Teixeira, Avelino V. (1); Liu, Shihui (1); Chopra, Arun; Singh, Yogendra; Sharma, Varsha; Leppla, Stephen H. (1)
CS (1) Oral Infection and Immunity Branch, NIDCR, NIH, Bethesda, MD, 20892 USA
SO IJMM International Journal of Medical Microbiology, (October, 2000) Vol. 290, No. 4-5, Supplement 30, pp. A58. print.
Meeting Info.: 9th European Workshop on Bacterial Protein Toxins Saint Maxime, France June 27-July 02, 1999
ISSN: 1438-4221.
DT Conference
LA English
SL English

L16 ANSWER 54 OF 123 USPATFULL
AN 1999:141912 USPATFULL
TI Compositions and methods for delivery of genetic material
IN Weiner, David B., Merion, PA, United States
Williams, William V., Havertown, PA, United States
Wang, Bin, Havertown, PA, United States
PA The Trustees of The University of Pennsylvania, Philadelphia, PA, United States (U.S. corporation)
The Wistar Institute, Philadelphia, PA, United States (U.S. corporation)
PI US 5981505 19991109
WO 9416737 19940804
AI US 1997-979385 19971126 (8)
WO 1994-US899 19940126
19950828 PCT 371 date
19950828 PCT 102(e) date
RLI Continuation-in-part of Ser. No. US 1993-124962, filed on 21 Sep 1993, now abandoned And a continuation-in-part of Ser. No. US 1993-93235, filed on 15 Jul 1993, now abandoned And a continuation of Ser. No. US 1995-495684, filed on 28 Aug 1995, now abandoned which is a continuation-in-part of Ser. No. US 1993-125012, filed on 21 Sep 1993, now patented, Pat. No. US 5593972, issued on 14 Jan 1997 which is a continuation-in-part of Ser. No. US 1993-29336, filed on 11 Mar 1993, now abandoned which is a continuation-in-part of Ser. No. US 1993-8342, filed on 26 Jan 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Railey, II, Johnny F.
LREP Woodcock Washburn Kurtz Mackiewicz & Norris LLP
CLMN Number of Claims: 75
ECL Exemplary Claim: 1
DRWN 23 Drawing Figure(s); 12 Drawing Page(s)
LN.CNT 4084
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 55 OF 123 USPATFULL
AN 1999:141305 USPATFULL
TI Adjuvant for transcutaneous immunization
IN Glenn, Gregory M., Bethesda, MD, United States

Alving, Carl R., Bethesda, MD, United States
PA The United States of America as represented by the U.S. Army Medical Research & Material Command, Washington, DC, United States (U.S. government)
PI US 5980898 19991109
AI US 1997-896085 19970717 (8)
RLI Continuation-in-part of Ser. No. US 1996-749164, filed on 14 Nov 1996
DT Utility
FS Granted
EXNAM Primary Examiner: Saunders, David; Assistant Examiner: Tung, Mary Beth
LREP Pillsbury, Madison & Sutro LLP
CLMN Number of Claims: 13
ECL Exemplary Claim: 1,11
DRWN 1 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 1988
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 56 OF 123 USPATFULL
AN 1999:109966 USPATFULL
TI Opsonin-enhanced cells, and methods of modulating an immune response to an antigen
IN Segal, Andrew H., Boston, MA, United States
PA Whitenead Institute for Biomedical Research, Cambridge, MA, United States (U.S. corporation)
PI US 5951976 19990914
AI US 1997-826259 19970327 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Hutzell, Paula K.; Assistant Examiner: Bansal, Geetha P.
LREP Banner & Witcoff, Ltd.
CLMN Number of Claims: 22
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 2180
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 57 OF 123 USPATFULL
AN 1999:65064 USPATFULL
TI Transdermal delivery system for antigen
IN Alving, Carl R., Bethesda, MD, United States
Glenn, Gregory M., Bethesda, MD, United States
PA The United States of America as represented by the Secretary of the Army, Washington, DC, United States (U.S. government)
PI US 5910306 19990608
AI US 1996-749164 19961114 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Saunders, David; Assistant Examiner: Tung, Mary Beth
LREP Pillsbury Madison & Sutro LLP
CLMN Number of Claims: 29
ECL Exemplary Claim: 1,10
DRWN No Drawings
LN.CNT 1154
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 58 OF 123 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 1999285696 EMBASE
TI **Anthrax protective antigen**: Prepore-to-pore conversion.
AU Miller C.J.; Elliott J.L.; Collier R.J.
CS R.J. Collier, 200 Longwood Ave., Boston, MA 02115, United States

SO Biochemistry, (10 Aug 1999) 38/32 (10432-10441).
Refs: 29
ISSN: 0006-2960 CODEN: BICHAW
CY United States
DT Journal; Article
FS 004 Microbiology
029 Clinical Biochemistry
LA English
SL English

L16 ANSWER 59 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 18
AN 1999:386505 BIOSIS
DN PREV199900386505
TI Autogenous regulation of the Bacillus **anthracis** pag operon.
AU Hoffmaster, Alex R.; Koehler, Theresa M. (1)
CS (1) Department of Microbiology and Molecular Genetics, University of Texas-Houston Health Science Center Medical School, 6431 Fannin St., JFB 1.765, Houston, TX, 77030 USA
SO Journal of Bacteriology, (Aug., 1999) Vol. 181, No. 15, pp. 4485-4492.
ISSN: 0021-9193.
DT Article
LA English
SL English

L16 ANSWER 60 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 19
AN 1999:338648 BIOSIS
DN PREV199900338648
TI **Anthrax** toxin entry into polarized epithelial cells.
AU Beauregard, Kathryn E.; Wimer-Mackin, Susan; Collier, R. John; Lencer, Wayne I. (1)
CS (1) GI Cell Biology, Children's Hospital, 300 Longwood Ave., Enders 1220, Boston, MA, 02115 USA
SO Infection and Immunity, (June, 1999) Vol. 67, No. 6, pp. 3026-3030.
ISSN: 0019-9567.
DT Article
LA English
SL English

L16 ANSWER 61 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 20
AN 1999:322259 BIOSIS
DN PREV199900322259
TI Disruption of **anthrax** toxin binding with the use of human antibodies and competitive inhibitors.
AU Cirino, Nick M.; Sblattero, Daniele; Allen, David; Peterson, Scott R.; Marks, James D.; Jackson, Paul J.; Bradbury, Andrew; Lehnert, Bruce E. (1)
CS (1) Los Alamos National Laboratory, Los Alamos, NM, 87545 USA
SO Infection and Immunity, (June, 1999) Vol. 67, No. 6, pp. 2957-2963.
ISSN: 0019-9567.
DT Article
LA English
SL English

L16 ANSWER 62 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 21
AN 1999:227788 BIOSIS
DN PREV199900227788
TI Identification of a **receptor**-binding region within domain 4 of the **protective antigen** component of **anthrax** toxin.

AU Varughese, Mini; Teixeira, Avelino V.; Liu, Shihui; Leppla, Stephen H. (1)
CS (1) Oral Infection and Immunity Branch, National Institute of Dental and Craniofacial Research, 30 Convent Dr. MSC 4350, Bldg. 30, Room 316, Bethesda, MD, 20892-4350 USA
SO Infection and Immunity, (April, 1999) Vol. 67, No. 4, pp. 1860-1865. ISSN: 0019-9567.
DT Article
LA English
SL English

L16 ANSWER 63 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC. DUPLICATE 22
AN 1999:227787 BIOSIS
DN PREV199900227787
TI Oligomerization of **anthrax** toxin **protective antigen** and binding of lethal factor during endocytic uptake into mammalian cells.
AU Singh, Yogendra; Klimpel, Kurt R.; Goel, Seema; Swain, Prabodha K.; Leppla, Stephen H. (1)
CS (1) Oral Infection and Immunity Branch, National Institute of Dental and Craniofacial Research, NIH, Bldg. 30, Rm. 309, Bethesda, MD, 20892 USA
SO Infection and Immunity, (April, 1999) Vol. 67, No. 4, pp. 1853-1859. ISSN: 0019-9567.
DT Article
LA English
SL English

L16 ANSWER 64 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC. DUPLICATE 23
AN 2000:23757 BIOSIS
DN PREV200000023757
TI **Anthrax** toxins.
AU Duesbery, N. S.; Vande Woude, G. F. (1)
CS (1) Division of Basic Sciences, NCI-FCRDC, Frederick, MD, 21702 USA
SO CMLS Cellular and Molecular Life Sciences, (Sept., 1999) Vol. 55, No. 12, pp. 1599-1609. ISSN: 1420-682X.
DT General Review
LA English
SL English

L16 ANSWER 65 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC. DUPLICATE 24
AN 1999:111784 BIOSIS
DN PREV199900111784
TI Functional analysis of the carboxy-terminal domain of Bacillus **anthracis protective antigen**.
AU Brossier, Fabien; Sirard, Jean-Claude; Guidi-Rontani, Chantal; Duflot, Edith; Mock, Michele (1)
CS (1) Unite Toxines Pathogenie Bacteriennes, Inst. Pasteur, 28 rue du Dr. Roux, 75724 Paris Cedex 15 France
SO Infection and Immunity, (Feb., 1999) Vol. 67, No. 2, pp. 964-967. ISSN: 0019-9567.
DT Article
LA English

L16 ANSWER 66 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC. DUPLICATE 25
AN 1999:263318 BIOSIS
DN PREV199900263318
TI Endoprotease PACE4 is Ca2+-dependent and temperature-sensitive and can partly rescue the phenotype of a furin-deficient cell strain.

AU Sucic, Joseph F. (1); Moehring, Joan M.; Inocencio, Noel M.; Luchini, Jason W.; Moehring, Thomas J.
CS (1) Biology Department, University of Michigan-Flint, 303 East Kearsley St., Flint, MI, 48502-1950 USA
SO Biochemical Journal, (May 1, 1999) Vol. 339, No. 3, pp. 639-647. ISSN: 0264-6021.
DT Article
LA English
SL English

L16 ANSWER 67 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1999:414748 BIOSIS
DN PREV199900414748
TI Expression and purification of the recombinant **protective antigen** of Bacillus **anthracis**.
AU Gupta, Pankaj; Waheed, S. M.; Bhatnagar, R. (1)
CS (1) Centre for Biotechnology, Jawaharlal Nehru University, New Delhi, 110067 India
SO Protein Expression and Purification, (Aug., 1999) Vol. 16, No. 3, pp. 369-376. ISSN: 1046-5928.
DT Article
LA English
SL English

L16 ANSWER 68 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC. DUPLICATE 26
AN 1999:87248 BIOSIS
DN PREV199900087248
TI Activation of phospholipase C and protein kinase C is required for expression of **anthrax** lethal toxin cytotoxicity in J774A.1 cells.
AU Bhatnagar, Rakesh (1); Goila, Nidhi Ahuja Ritu; Batra, Smriti; Waheed, S. M.; Gupta, Pankaj
CS (1) Centre Biotechnol., Jawaharlal Nehru Univ., New Delhi-110 067 India
SO Cellular Signalling, (Feb., 1999) Vol. 11, No. 2, pp. 111-116. ISSN: 0898-6568.
DT Article
LA English

L16 ANSWER 69 OF 123 LIFESCI COPYRIGHT 2002 CSA
AN 2000:14130 LIFESCI
TI Mechanism of membrane translocation by **anthrax** toxin: Insertion and pore formation by **protective antigen**
AU Collier, R.J.
CS Microbiology and Molecular Genetics, Harvard Medical School, 200 Longwood Avenue, Boston, MA 02115, USA
SO Journal of Applied Microbiology, (19990800) vol. 87, no. 2, 283. Meeting Info.: 3rd International Conference on Anthrax. Plymouth (UK). 7-10 Sep 1998. ISSN: 1364-5072.
DT Journal
TC Dictionary
FS X; J
LA English
SL English

L16 ANSWER 70 OF 123 LIFESCI COPYRIGHT 2002 CSA
AN 2000:40949 LIFESCI
TI **Anthrax** toxin fusion proteins for intracellular delivery of macromolecules
AU Leppla, S.H.; Arora, N.; Varughese, M.

CS Oral Infection and Immunity Branch, National Institute of Dental Research, NIH, Bethesda, MD 20892, USA
SO Journal of Applied Microbiology, (19990800) vol. 87, no. 2, 284. Meeting Info.: 3rd International Conference on Anthrax. Plymouth (UK). 7-10 Sep 1998. ISSN: 1364-5072.
DT Journal
TC Abstract
FS J; V; W3
LA English
SL English

L16 ANSWER 71 OF 123 CAPLUS COPYRIGHT 2002 ACS
AN 1999:27954 CAPLUS
DN 130:77075
TI Targetting and uptake of DNA by animal cells by **receptor** -mediated endocytosis using fusion protein of toxins and DNA-binding proteins
IN Grandi, Guido
PA Chiron S.P.A., Italy
SO PCT Int. Appl., 85 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9859065	A1	19981230	WO 1998-IB1005	19980618

W: JP, US
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
PRAI GB 1997-13122 19970620
RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 72 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 27
AN 1999:246978 BIOSIS
DN PREV199900246978
TI Purification of the **protective antigen** from Bacillus **anthracis**.
AU Cho, Soung-Kun; Park, Jeung-Moon; Choi, Young-Keel; Kim, Seong-Joo; Chai, Young-Gyu (1)
CS (1) Department of Biochemistry and Molecular Biology, Hanyang University, Ansan, Kyunggi-do, 425-791 South Korea
SO Journal of the Korean Society for Microbiology, (Dec., 1998) Vol. 33, No. 6, pp. 589-594. ISSN: 0253-3162.
DT Article
LA Korean
SL English

L16 ANSWER 73 OF 123 CAPLUS COPYRIGHT 2002 ACS
AN 1999:37101 CAPLUS
DN 130:233465
TI Activation of phospholipase C and protein kinase C is required for expression of **anthrax** lethal toxin cytotoxicity in J774A.1 cells
AU Bhatnagar, Rakesh; Ahuja, Nidhi; Goila, Ritu; Batra, Smriti; Waheed, S. M.; Gupta, Pankaj
CS Centre for Biotechnology, Jawaharlal Nehru University, New Delhi, 110 067, India
SO Cellular Signalling (1998), Volume Date 1999, 11(2), 111-116

CODEN: CESIEY; ISSN: 0898-6568
PB Elsevier Science Inc.
DT Journal
LA English
RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 74 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 28
AN 1998:228710 BIOSIS
DN PREV199800228710
TI Internalization of a Bacillus **anthracis protective antigen**-c-Myc fusion protein mediated by cell surface anti-c-Myc antibodies.
AU Varughese, Mini; Chi, Angela; Teixeira, Avelino V.; Nicholls, Peter J.; Keith, Jerry M.; Leppla, Stephen H. (1)
CS (1) Oral Infect. Immunity Branch, Natl. Inst. Dent. Res., Build. 30, Room 316, 30 Convent Dr. MSC 4350, Bethesda, MD 20892-4350 USA
SO Molecular Medicine (New York), (Feb., 1998) Vol. 4, No. 2, pp. 87-95. ISSN: 1076-1551.
DT Article
LA English

L16 ANSWER 75 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1998:459114 BIOSIS
DN PREV199800459114
TI Redirecting **anthrax** toxin **protective antigen** to new receptors to create cell-type specific cytotoxic and therapeutic agents.
AU Varughese, M.; Teixeira, A.; Chi, A.; Nicholls, P.; Keith, J.; Leppla, S.
CS Natl. Inst. Dent. Res., Natl. Inst. Health, Build. 30, Bethesda, MD 20892-4350 USA
SO Zentralblatt fuer Bakteriologie Supplement, (1998) Vol. 29, pp. 76-77. Meeting Info.: Eighth European Workshop on Bacterial Protein Toxins Staffelstein, Kloster Banz, Germany June 29-July 4, 1997 ISSN: 0941-018X.
DT Conference
LA English

L16 ANSWER 76 OF 123 USPATFULL
AN 97:94207 USPATFULL
TI **Anthrax** toxin fusion proteins and related methods
IN Leppla, Stephen H., Bethesda, MD, United States
Klimpel, Kurt R., Gaithersburg, MD, United States
Arora, Naveen, Delhi, India
Singh, Yogendra, Delhi, India
Nichols, Peter J., Welling Kent, United Kingdom
PA The Government of the United States as represented by the Secretary of the Department of Health and Human Services, Washington, DC, United States (U.S. government)
PI US 5677274 19971014
AI US 1993-82849 19930625 (8)
RLI Continuation-in-part of Ser. No. US 1993-21601, filed on 12 Feb 1993, now patented, Pat. No. US 5591631
DT Utility
FS Granted
EXNAM Primary Examiner: Jagannathan, Vasu S.; Assistant Examiner: Romeo, David S.
LREP Townsend and Townsend and Crew
CLMN Number of Claims: 12
ECL Exemplary Claim: 1
DRWN 1 Drawing Figure(s); 1 Drawing Page(s)

LN.CNT 3382
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 77 OF 123 USPATFULL
AN 97:14677 USPATFULL
TI Methods and reagents for inhibiting furin endoprotease
IN Thomas, Gary, Tualatin, OR, United States
Anderson, Eric D., Portland, OR, United States
Thomas, Laurel, Tualatin, OR, United States
Hayflick, Joel S., Seattle, WA, United States
PA State of Oregon, Acting by and through the Oregon State Board of Higher Education on Behalf of the Oregon Health Sciences University, a non-profit organization, Portland, OR, United States (U.S. corporation)
PI US 5604201 19970218
AI US 1993-2202 19930108 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Lau, Kawai
LREP Banner & Allegretti, Ltd.
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 6 Drawing Figure(s); 6 Drawing Page(s)
LN.CNT 1307
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 78 OF 123 USPATFULL
AN 97:1356 USPATFULL
TI **Anthrax** toxin fusion proteins, nucleic acid encoding same
IN Leppla, Stephen H., Bethesda, MD, United States
Klimpel, Kurt R., Gaithersburg, MD, United States
Arora, Naveen, Delhi, India
Singh, Yogendra, Delhi, India
Nicholls, Peter J., Welling Kent, United Kingdom
PA The United States of America as represented by the Department of Health and Human Services, Washington, DC, United States (U.S. government)
PI US 5591631 19970107
AI US 1993-21601 19930212 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Walsh, Stephen G.
LREP Townsend and Townsend and Crew
CLMN Number of Claims: 13
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 2181
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 79 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 29
AN 1997:158460 BIOSIS
DN PREV199799457663
TI Crystal structure of the **anthrax** toxin **protective antigen**.
AU Petosa, Carlo (1); Collier, R. John; Klimpel, Kurt R.; Leppla, Stephen H.; Liddington, Robert C.
CS (1) Biochemistry Dep., Univ. Leicester, Leicester LE1 7RH UK
SO Nature (London), (1997) Vol. 385, No. 6619, pp. 833-838.
ISSN: 0028-0836.
DT Article
LA English

L16 ANSWER 80 OF 123 CAPLUS COPYRIGHT 2002 ACS

AN 1998:419479 CAPLUS
DN 129:199150
TI Secondary structure and lipid binding of **anthrax** lethal and edema toxin proteins of B. **anthracis**
AU Wang, X. M.; Mock, M.; Ruysscaert, J. M.; Cabiaux, V.
CS Universite Libre de Bruxelles, Brussels, 1050, Belg.
SO Zentralblatt fuer Bakteriologie, Supplement (1997), 29(Bacterial Protein Toxins), 144-145
CODEN: ZBASE2; ISSN: 0941-018X
PB Gustav Fischer Verlag
DT Journal
LA English

L16 ANSWER 81 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 30
AN 1997:350041 BIOSIS
DN PREV199799649244
TI Elucidation of functionally active domains in the molecules of **protective antigen** Bacillus **anthracis** toxin.
AU Noskov, A. N.; Kravchenko, T. B.; Noskova, V. P.
CS State Res. Cent. Appl. Microbiol., Obolensk Russia
SO Vestnik Rossiiskoi Akademii Meditsinskikh Nauk, (1997) Vol. 0, No. 6, pp. 20-24.
ISSN: 0869-6047.
DT Article
LA Russian
SL English

L16 ANSWER 82 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1997:333380 BIOSIS
DN PREV199799632583
TI Isolation and characterization of Chinese hamster ovary cell mutants lacking the **receptor** for **anthrax** toxin **protective antigen.**
AU Leppla, S. H.; Gu, M. L.; Gordon, V. M.; Arora, N.; Singh, Y.; Klimpel, K. R.
CS Lab. Microbial Ecol., National Inst. Dental Res., National Inst. Health, Bethesda, MD 20892-4350 USA
SO Zentralblatt fuer Bakteriologie Supplement, (1996) Vol. 28, No. 0, pp. 119-120.
Meeting Info.: Seventh European Workshop on Bacterial Protein Toxins Hindsgavl, Denmark July 2-7, 1995
ISSN: 0941-018X.
DT Book; Conference
LA English

L16 ANSWER 83 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 31
AN 1996:221226 BIOSIS
DN PREV199698777355
TI Characterization of lethal factor binding and cell **receptor** binding domains of **protective antigen** of Bacillus **anthracis** using monoclonal antibodies.
AU Little, Stephen F. (1); Novak, Jeanne M. (1); Lowe, John R. (1); Leppla, Stephen H. (1); Singh, Yogendra; Klimpel, Kurt R.; Lidgerding, Burton C. (1); Friedlander, Arthur M. (1)
CS (1) US Army Med. Res., Inst. Infectious Diseases, Fort Detrick, Frederick, MD 21702-5011 USA
SO Microbiology (Reading), (1996) Vol. 142, No. 3, pp. 707-715.
ISSN: 1350-0872.
DT Article
LA English

L16 ANSWER 84 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1996:305967 BIOSIS
DN PREV199699028323
TI Binding and uptake of **anthrax** toxin components and fusion proteins by eukaryotic cells.
AU Leppla, S. H.; Klimpel, K. R.; Gordon, V. M.; Arora, N.; Singh, Y.
CS Lab. Microbiol. Ecol., Natl. Inst. Dent. Res., NIH, Bethesda, MD 20892 USA
SO Toxicon, (1996) Vol. 34, No. 3, pp. 296.
Meeting Info.: Fifth Pan American Symposium on Animal, Plant and Microbial Toxins Frederick, Maryland, USA July 30-August 4, 1995
ISSN: 0041-0101.
DT Conference
LA English

L16 ANSWER 85 OF 123 MEDLINE
AN 97141282 MEDLINE
DN 97141282 PubMed ID: 8987626
TI Thermostabilization of **protective antigen**--the binding component of **anthrax** lethal toxin.
AU Radha C; Salotra P; Bhat R; Bhatnagar R
CS Centre for Biotechnology, Jawaharlal Nehru University, New Delhi, India.
SO JOURNAL OF BIOTECHNOLOGY, (1996 Oct 1) 50 (2-3) 235-42.
Journal code: 8411927. ISSN: 0168-1656.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Biotechnology
EM 199702
ED Entered STN: 19970227
Last Updated on STN: 19970227
Entered Medline: 19970213

L16 ANSWER 86 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 32
AN 1996:122757 BIOSIS
DN PREV199698694892
TI Expression and purification of **anthrax** toxin **protective antigen** from Escherichia coli.
AU Sharma, Manju (1); Swain, Prabodha K. (1); Chopa, Arun P. (1); Chaudhary, Vijay K.; Singh, Yogendra
CS (1) Genetic Eng. Div., Centre Biochem. Technol., Mall Road, Delhi 110 007 India
SO Protein Expression and Purification, (1996) Vol. 7, No. 1, pp. 33-38.
ISSN: 1046-5928.
DT Article
LA English

L16 ANSWER 87 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 33
AN 1996:514796 BIOSIS
DN PREV199699237152
TI Detection of functional domains in the molecule of **protective antigen** of Bacillus **anthracis** toxin.
AU Noskov, A. N.; Kravchenko, T. B.; Koskova, V. P.
CS State Res. Cent. Appl. Microbiol., Obolensk Russia
SO Molekulyarnaya Genetika Mikrobiologiya i Virusologiya, (1996) Vol. 0, No. 3, pp. 16-20.
ISSN: 0208-0613.
DT Article
LA Russian
SL English

L16 ANSWER 88 OF 123 LIFESCI COPYRIGHT 2002 CSA
AN 96:34906 LIFESCI
TI Similarities between the lethal factor of Bacillus **anthracis** and leukotreine A sub(4) hydrolase
AU Menard, A.; Mock, M.; Montecucco, C.
CS Centro CNR Biomembrane Dipartimento di Scienze Biomediche, Universita di Padova, Via Trieste 75, 35121 Padova, Italy
SO MOL. MICROBIOL., (1995) vol. 18, no. 5, pp. 991-992.
ISSN: 0950-382X.
DT Journal
FS J; X
LA English

L16 ANSWER 89 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 34
AN 1995:203456 BIOSIS
DN PREV199598217756
TI **Protective antigen**-binding domain of **anthrax** lethal factor mediates translocation of a heterologous protein fused to its amino- or carboxy-terminus.
AU Milne, Jill C.; Blanke, Steven R.; Hanna, Philip C.; Collier, R. John (1)
CS (1) Dep. Microbiol. Mol. Genetics, Shipley Inst. Med., Harvard Med. Sch., Boston, MA 02115 USA
SO Molecular Microbiology, (1995) Vol. 15, No. 4, pp. 661-666.
ISSN: 0950-382X.
DT Article
LA English

L16 ANSWER 90 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1995:149035 BIOSIS
DN PREV199598163335
TI Redirecting **anthrax** toxin to HIV infected cells.
AU Teixeira, A. V.; Leppla, S. H.
CS Lab. Microbial Ecol., NIDR, NIH, Bethesda, MD 20892 USA
SO AMERICAN SOCIETY FOR MICROBIOLOGY.. (1995) pp. 69. Human retroviruses and related infections.
Publisher: American Society for Microbiology (ASM) Books Division, 1325 Massachusetts Ave. NW, Washington, DC 20005-4171, USA.
Meeting Info.: 2nd National Conference Washington, D.C., USA January 29-February 2, 1995
ISBN: 1-55581-097-7.
DT Conference
LA English

L16 ANSWER 91 OF 123 CAPLUS COPYRIGHT 2002 ACS
AN 1994:696634 CAPLUS
DN 121:296634
TI Lyophilized ligand-**receptor** complexes for assays and sensors
IN Ligler, Frances S.; Whelan, James P.
PA United States Dept. of the Navy, USA; U.S. Drug Testing, Inc.
SO U.S., 14 pp.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 5354654	A	19941011	US 1993-92518	19930716
	WO 9502703	A1	19950126	WO 1994-US7806	19940715

W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, ES, FI, GB, GE, HU, JP, KE, KG, KP, KR, KZ, LK, LT, LU, LV, MD, MG, MN, MW,

NL, NO, NZ, PL, PT, RO, RU, SD, SE, SI, SK, TJ, TT, UA, UZ, VN
RW: KE, MW, SD, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

CA	2167275	AA	19950126	CA 1994-2167275	19940715
AU	9473603	A1	19950213	AU 1994-73603	19940715
AU	685148	B2	19980115		
EP	710293	A1	19960508	EP 1994-922533	19940715

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE

PRAI	US 1993-92518	19930716	
	WO 1994-US7806	19940715	

L16 ANSWER 92 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1994:421456 BIOSIS
DN PREV199497434456
TI **Anthrax** toxin mechanisms of **receptor** binding and internalization.
AU Leppla, Stephen H.; Klimpel, Kurt R.; Arora, Naveen
CS Lab. Microbial Ecol., Natl. Inst. Dent. Res., Natl. Inst. Health, Bethesda, MD 20892 USA
SO Kado, C. I. [Editor]; Crosa, J. H. [Editor]. Developments in Plant Pathology, (1994) Vol. 3, pp. 127-139. Developments in Plant Pathology; Molecular mechanisms of bacterial virulence.
Publisher: Kluwer Academic Publishers PO Box 989, 3300 AZ Dordrecht, Netherlands.
Meeting Info.: Conference South Lake Tahoe, California, USA September 10-13, 1992
ISBN: 0-7923-1901-X.
DT Book; Conference
LA English

L16 ANSWER 93 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 35
AN 1995:35523 BIOSIS
DN PREV199598049823
TI The chymotrypsin-sensitive site, FFD-315, in **anthrax** toxin **protective antigen** is required for translocation of lethal factor.
AU Singh, Yogendra; Klimpel, Kurt R. (1); Arora, Naveen (1); Sharma, Manju; Leppla, Stephen H. (1)
CS (1) Lab. Microbial Ecol., Natl. Inst. Dental Res., Build. 30, Room 309, NIH, Bethesda, MD 20892 USA
SO Journal of Biological Chemistry, (1994) Vol. 269, No. 46, pp. 29039-29046.
ISSN: 0021-9258.
DT Article
LA English

L16 ANSWER 94 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 36
AN 1994:554245 BIOSIS
DN PREV199598013793
TI Cytotoxic Effects of a Chimeric Protein Consisting of Tetanus Toxin Light Chain and **Anthrax** Toxin Lethal Factor in Non-neuronal Cells.
AU Arora, Naveen; Williamson, Lura C.; Leppla, Stephen H.; Halpern, Jane L. (1)
CS (1) Build. 29 Room 103, 8800 Rockville Pike, Bethesda, MD 20892 USA
SO Journal of Biological Chemistry, (1994) Vol. 269, No. 42, pp. 26165-26171.
ISSN: 0021-9258.
DT Article
LA English

L16 ANSWER 95 OF 123 CAPLUS COPYRIGHT 2002 ACS
AN 1994:475769 CAPLUS

DN 121:75769
TI Protein synthesis is required for expression of **anthrax** lethal toxin cytotoxicity
AU Bhatnagar, R.; Friedlander, A. M.
CS U.S. Army Medical Research Inst. Infectious Diseases, Frederick, MD, 21702-5011, USA
SO Infect. Immun. (1994), 62(7), 3958-62
CODEN: INFIBR; ISSN: 0019-9567
DT Journal
LA English

L16 ANSWER 96 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 37
AN 1994:347356 BIOSIS
DN PREV199497360356
TI Protein synthesis is required for expression of **anthrax** lethal toxin cytotoxicity.
AU Bhatnagar, R.; Friedlander, A. M. (1)
CS (1) United States Army Med. Res. Inst. Infectious Dis., Frederick, MD 21702-5011 USA
SO Infection and Immunity, (1994) Vol. 62, No. 7, pp. 2958-2962.
ISSN: 0019-9567.
DT Article
LA English

L16 ANSWER 97 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 38
AN 1994:216366 BIOSIS
DN PREV199497229366
TI The effects of pH on the interaction of **anthrax** toxin lethal and edema factors with phospholipid vesicles.
AU Kochi, Sims K.; Martin, Isabelle; Schiavo, Giampietro; Mock, Michele; Cabiaux, Veronique (1)
CS (1) Lab. Chem. Physique Macromolecules aux Interfaces, Universite de Bruxelles, CP 206/2, Boulevard du Triomphe, 1050 Bruxelles Belgium
SO Biochemistry, (1994) Vol. 33, No. 9, pp. 2604-2609.
ISSN: 0006-2960.
DT Article
LA English

L16 ANSWER 98 OF 123 CAPLUS COPYRIGHT 2002 ACS
AN 1994:595062 CAPLUS
DN 121:195062
TI Development of **anthrax**-toxin based fusion proteins for targeting of HIV-1-infected cells
AU Leppla, S. H.; Klimpel, K. R.; Arora, N.
CS Laboratory of Microbial Ecology, National Institute of Dental Research, Bethesda, MD, 20892, USA
SO Zentralbl. Bakteriol., Suppl. (1994), 24(Bacterial Protein Toxins), 431-42
CODEN: ZBASE2; ISSN: 0941-018X
DT Journal
LA English

L16 ANSWER 99 OF 123 CAPLUS COPYRIGHT 2002 ACS
AN 1994:597701 CAPLUS
DN 121:197701
TI **Anthrax** toxin mechanisms of **receptor** binding and internalization
AU Leppla, Stephen H.; Klimpel, Kurt R.; Arora, Naveen
CS Lab. Microbial Ecol., Nat. Inst. Dental Res., Bethesda, MD, USA
SO Dev. Plant Pathol. (1994), 3(Molecular Mechanisms of Bacterial Virulence), 127-39

CODEN: DPPAEF
DT Journal; General Review
LA English

L16 ANSWER 100 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 39
AN 1994:227429 BIOSIS
DN PREV199497240429
TI The channel formed in planar lipid bilayers by the **protective antigen** component of **anthrax** toxin.
AU Finkelstein, Alan
CS Deps. Physiology Biophysics, Albert Einstein College Medicine, 1300 Morris Park Ave., Bronx, NY 10461 USA
SO Toxicology, (1994) Vol. 87, No. 1-3, pp. 29-41.
ISSN: 0300-483X.
DT General Review
LA English

L16 ANSWER 101 OF 123 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 93353907 EMBASE
DN 1993353907
TI Characterization of Clostridium perfringens iota-toxin genes and expression in Escherichia coli.
AU Perelle S.; Gibert M.; Boquet P.; Popoff M.R.
CS Laboratoire des Toxines Microbiennes, Institut Pasteur, 28 Rue du Dr. Roux,75724 Paris Cedex 15, France
SO Infection and Immunity, (1993) 61/12 (5147-5156).
ISSN: 0019-9567 CODEN: INFIBR
CY United States
DT Journal; Article
FS 004 Microbiology
LA English
SL English

L16 ANSWER 102 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 40
AN 1993:165889 BIOSIS
DN PREV199395086939
TI Residues 1-254 of **anthrax** toxin lethal factor are sufficient to cause cellular uptake of fused polypeptides.
AU Arora, Naveen; Leppla, Stephen H. (1)
CS (1) Lab. Microbial Ecol., NIDR, Building 30, Room 309, NIH, Bethesda, MD 20892 USA
SO Journal of Biological Chemistry, (1993) Vol. 268, No. 5, pp. 3334-3341.
ISSN: 0021-9258.
DT Article
LA English

L16 ANSWER 103 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1993:142483 BIOSIS
DN PREV199395075283
TI Characterization of macrophage sensitivity and resistance to **anthrax** lethal toxin.
AU Friedlander, Arthur M. (1); Bhatnagar, Rakesh; Leppla, Stephen H.; Johnson, Larry; Singh, Yogendra
CS (1) U.S. Army Med. Res. Inst. Infectious Diseases, Frederick, MD 21702-5011 USA
SO Infection and Immunity, (1993) Vol. 61, No. 1, pp. 245-252.
ISSN: 0019-9567.
DT Article
LA English

L16 ANSWER 104 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 41
AN 1992:478051 BIOSIS
DN BA94:109426
TI FUNCTIONAL CHARACTERIZATION OF PROTEASE-TREATED BACILLUS-**ANTHRACIS PROTECTIVE ANTIGEN**.
AU NOVAK J M; STEIN M-P; LITTLE S F; LEPPLA S H; FRIEDLANDER A M
CS BACTERIOLOGY DIVISION, UNITED STATES ARMY MEDICAL RESEARCH INSTITUTE INFECTIOUS DISEASES, FORT DETRICK, FREDERICK, MD. 21702-5011.
SO J BIOL CHEM, (1992) 267 (24), 17186-17193.
CODEN: JBCHA3. ISSN: 0021-9258.
FS BA; OLD
LA English

L16 ANSWER 105 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 42
AN 1992:430454 BIOSIS
DN BA94:82579
TI FUSIONS OF **ANTHRAX** TOXIN LETHAL FACTOR TO THE ADP-RIBOSYLATION DOMAIN OF PSEUDOMONAS EXOTOXIN A ARE POTENT CYTOTOXINS WHICH ARE TRANSLOCATED TO THE CYTOSOL OF MAMMALIAN CELLS.
AU ARORA N; KLIMEPL K R; SINGH Y; LEPPLA S H
CS LABORATORY MICROBIAL ECOLOGY, NATIONAL INSTITUTE DENTAL RESEARCH, BLDG. 30, ROOM 309, NIH, BETHESDA, MD. 20892.
SO J BIOL CHEM, (1992) 267 (22), 15542-15548.
CODEN: JBCHA3. ISSN: 0021-9258.
FS BA; OLD
LA English

L16 ANSWER 106 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 43
AN 1993:75616 BIOSIS
DN PREV199395040116
TI **Anthrax** toxin **protective antigen** is activated by a cell surface protease with the sequence specificity and catalytic properties of furin.
AU Klimpel, Kurt R.; Molloy, Sean S.; Thomas, Gary; Leppla, Stephen H. (1)
CS (1) Bldg. 30, Room 309, Natl. Inst. Health, Bethesda, Md. 20892 USA
SO Proceedings of the National Academy of Sciences of the United States of America, (1992) Vol. 89, No. 21, pp. 10277-10281.
ISSN: 0027-8424.
DT Article
LA English

L16 ANSWER 107 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 44
AN 1992:359590 BIOSIS
DN BR43:37740
TI **ANTHRAX PROTECTIVE ANTIGEN RECEPTOR**
IDENTIFICATION OF A **PROTECTIVE ANTIGEN** BINDING PROTEIN BY CHEMICAL CROSS-LINKING.
AU FRIEDLANDER A M; RAZIUDDIN A
CS U.S. ARMY MEDICAL RESEARCH INSTITUTE INFECTIOUS DISEASES, FREDERICK, MD. 21702, USA.
SO WITHOLT, B., ET AL. (ED.). ZENTRALBLATT FUER BAKTERIOLOGIE SUPPLEMENT, 23; (INTERNATIONAL JOURNAL OF MEDICAL MICROBIOLOGY SUPPLEMENT, 23). BACTERIAL PROTEIN TOXINS; FIFTH EUROPEAN WORKSHOP, VELDHOVEN, NETHERLANDS, JUNE 30-JULY 5, 1991. XIV+513P. GUSTAV FISCHER VERLAG: STUTTGART, GERMANY; NEW YORK, NEW YORK, USA. ILLUS. (1992) 0 (0), 365-366.
CODEN: ZBASE2. ISBN: 3-437-11421-2, 1-56081-342-3.
DT Conference
FS BR; OLD

LA English

L16 ANSWER 108 OF 123 CAPLUS COPYRIGHT 2002 ACS
AN 1993:423097 CAPLUS
DN 119:23097
TI Location of **receptor**-binding region of **protective antigen** from Bacillus **anthracis**
AU Little, S. F.; Lowe, J. R.
CS Army Med. Res. Inst. Infect. Dis., Fort Detrick, MD, USA
SO Report (1991), Order No. AD-A242 794, 15 pp. Avail.: NTIS From: Gov. Rep. Announce. Index (U. S.) 1992, 92(5), Abstr. No. 211,634
DT Report
LA English

L16 ANSWER 109 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC. DUPLICATE 45
AN 1992:27197 BIOSIS
DN BA93:16472
TI FUNCTIONAL MAPPING OF **ANTHRAX** TOXIN LETHAL FACTOR BY IN-FRAME INSERTION MUTAGENESIS.
AU QUINN C P; SINGH Y; KLIMPEL K R; LEPPLA S H
CS LAB. MICROBIAL ECOL., NATIONAL INST. DENTAL RES., BLDG. 30, RM. 309, NATIONAL INST. HEALTH, BETHESDA, MD. 20892-0300.
SO J BIOL CHEM, (1991) 266 (30), 20124-20130.
CODEN: JBCHA3. ISSN: 0021-9258.
FS BA; OLD
LA English

L16 ANSWER 110 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC. DUPLICATE 46
AN 1991:461647 BIOSIS
DN BA92:106427
TI THE CARBOXYL-TERMINAL END OF **PROTECTIVE ANTIGEN** IS REQUIRED FOR **RECEPTOR** BINDING AND **ANTHRAX** TOXIN ACTIVITY.
AU SINGH Y; KLIMPEL K R; QUINN C P; CHAUDHARY V K; LEPPLA S H
CS LAB. MICROBIAL ECOL., NATL. INST. DENTAL RES., BLDG. 30, ROOM 309, NATL. INST. HEALTH, BETHESDA, MD. 20892-0300.
SO J BIOL CHEM, (1991) 266 (23), 15493-15497.
CODEN: JBCHA3. ISSN: 0021-9258.
FS BA; OLD
LA English

L16 ANSWER 111 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC. DUPLICATE 47
AN 1991:524876 BIOSIS
DN BA92:136336
TI **ANTHRAX PROTECTIVE ANTIGEN** INTERACTS WITH A SPECIFIC **RECEPTOR** ON THE SURFACE OF CHO-K1 CELLS.
AU ESCUYER V; COLLIER R J
CS DEP. MICROBIOL. MOL. GENETICS SHIPLEY INST. MED., HARVARD MED. SCH., 200 LONGWOOD AVE., BOSTON, MASS. 02215.
SO INFECT IMMUN, (1991) 59 (10), 3381-3386.
CODEN: INFIBR. ISSN: 0019-9567.
FS BA; OLD
LA English

L16 ANSWER 112 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC. DUPLICATE 48
AN 1992:9274 BIOSIS
DN BA93:9274
TI LOCATION OF **RECEPTOR**-BINDING REGION OF **PROTECTIVE**

ANTIGEN FROM BACILLUS-**ANTHRACIS**.
AU LITTLE S F; LOWE J R
CS U.S. ARMY MED. RES. INST. INFECTIOUS DISEASES, BACTERIOLOGY DIV., FORT DETRICK, FREDERICK, MD. 21702.
SO BIOCHEM BIOPHYS RES COMMUN, (1991) 180 (2), 531-537.
CODEN: BBRCA9. ISSN: 0006-291X.
FS BA; OLD
LA English

L16 ANSWER 113 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1991:381124 BIOSIS
DN BR41:53514
TI **ANTHRAX PROTECTIVE ANTIGEN RECEPTOR**
IDENTIFICATION OF A **PROTECTIVE ANTIGEN** BINDING PROTEIN BY CHEMICAL CROSS-LINKING.
AU RAZIUDDIN A; FRIEDLANDER A
CS U.S. ARMY MED. RES. INST. INFECT. DIS., FORT DETRICK, FREDERICK, MD. 21702-5011, USA.
SO 91ST GENERAL MEETING OF THE AMERICAN SOCIETY FOR MICROBIOLOGY 1991, DALLAS, TEXAS, USA, MAY 5-9, 1991. ABSTR GEN MEET AM SOC MICROBIOL. (1991) 91 (0), 75.
CODEN: AGMME8.
DT Conference
FS BR; OLD
LA English

L16 ANSWER 114 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 49
AN 1990:354468 BIOSIS
DN BA90:51047
TI PRODUCTION AND CHARACTERIZATION OF MONOCLONAL ANTIBODIES AGAINST THE LETHAL FACTOR COMPONENT OF BACILLUS-**ANTHRACIS** LETHAL TOXIN.
AU LITTLE S F; LEPPLA S H; FRIEDLANDER A M
CS U.S. ARMY MEDICAL RESEARCH INSTITUTE INFECTIOUS DISEASES, FORT DETRICK, FREDERICK, MD. 21701-5011.
SO INFECT IMMUN, (1990) 58 (6), 1606-1613.
CODEN: INFIBR. ISSN: 0019-9567.
FS BA; OLD
LA English

L16 ANSWER 115 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1991:44239 BIOSIS
DN BR40:21219
TI CHYMOTRYPSIN FRAGMENTS OF BACILLUS-**ANTHRACIS PROTECTIVE ANTIGEN** BIND TO RECEPTORS BIND LETHAL FACTOR AND UNDERGO **RECEPTOR**-MEDIATED ENDOCYTOSIS BUT DO NOT KILL J774A. 1 CELLS.
AU NOVAK J M; STEIN M P; FRIEDLANDER A M
CS U.S. ARMY MED. RES. INST. INFECT. DIS., BACTERIOL. DIV., FORT DETRICK, FREDERICK, MD. 21702-5011.
SO THIRTIETH ANNUAL MEETING OF THE AMERICAN SOCIETY FOR CELL BIOLOGY, SAN DIEGO, CALIFORNIA, USA, DECEMBER 9-13, 1990. J CELL BIOL. (1990) 111 (5 PART 2), 192A.
CODEN: JCLBA3. ISSN: 0021-9525.
DT Conference
FS BR; OLD
LA English

L16 ANSWER 116 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 50
AN 1990:48442 BIOSIS
DN BA89:25806
TI A DELETED VARIANT OF BACILLUS-**ANTHRACIS PROTECTIVE**

ANTIGEN IS NON-TOXIC AND BLOCKS **ANTHRAX** TOXIN ACTION IN-VIVO.
AU SINGH Y; CHAUDHARY V K; LEPPLA S H
CS BACTERIOL. DIV., U.S. ARMY MED. RES. INST. OF INFECTIOUS DISEASES, FORT DETRICK, FREDERICK, MD. 21701-5011.
SO J BIOL CHEM, (1989) 264 (32), 19103-19107.
CODEN: JBCHA3. ISSN: 0021-9258.
FS BA; OLD
LA English

L16 ANSWER 117 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 51
AN 1989:382594 BIOSIS
DN BA88:63184
TI INTERNALIZATION AND PROCESSING OF BACILLUS-**ANTHRACIS** LETHAL TOXIN BY TOXIN-SENSITIVE AND TOXIN-RESISTANT CELLS.
AU SINGH Y; LEPPLA S H; BHATNAGAR R; FRIEDLANDER A M
CS UNITED STATES ARMY MED. RES. INST. INFECTIOUS DISEASES, FORT DETRICK, FREDERICK, MD. 21701-5011.
SO J BIOL CHEM, (1989) 264 (19), 11099-11102.
CODEN: JBCHA3. ISSN: 0021-9258.
FS BA; OLD
LA English

L16 ANSWER 118 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 52
AN 1989:269617 BIOSIS
DN BA88:5699
TI **ANTHRAX** TOXIN CHANNEL-FORMING ACTIVITY OF **PROTECTIVE ANTIGEN** IN PLANAR PHOSPHOLIPID BILAYERS.
AU BLAUSTEIN R O; KOEHLER T M; COLLIER R J; FINKELSTEIN A
CS DEP. PHYSIOLOGY BIOPHYS., ALBERT EINSTEIN COLL. MED., 1300 MORRIS PARK AVENUE, BRONX, N.Y. 10461.
SO PROC NATL ACAD SCI U S A, (1989) 86 (7), 2209-2213.
CODEN: PNASA6. ISSN: 0027-8424.
FS BA; OLD
LA English

L16 ANSWER 119 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1989:374526 BIOSIS
DN BR37:53649
TI DELETION OF A TRYPSIN CLEAVAGE SITE OF **PROTECTIVE ANTIGEN** PROTEIN INACTIVATES **ANTHRAX** TOXIN.
AU SINGH Y; LEPPLA S H
CS U.S. ARMY MED. RES. INST. INFECTIOUS DISEASES, FREDERICK, MD. 21701-5011.
SO 89TH ANNUAL MEETING OF THE AMERICAN SOCIETY FOR MICROBIOLOGY, NEW ORLEANS, LOUISIANA, USA, MAY 14-18, 1989. ABSTR ANNU MEET AM SOC MICROBIOL. (1989) 89 (0), 64.
CODEN: ASMACK. ISSN: 0094-8519.
DT Conference
FS BR; OLD
LA English

L16 ANSWER 120 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1988:397118 BIOSIS
DN BA86:69757
TI PRODUCTION AND CHARACTERIZATION OF MONOCLONAL ANTIBODIES TO THE **PROTECTIVE ANTIGEN** COMPONENT OF BACILLUS-**ANTHRACIS** TOXIN.
AU LITTLE S F; LEPPLA S H; CORA E
CS U.S. ARMY MED. RES. INST. INFECTIOUS DISEASES, FORT DETRICK, FREDERICK, MD. 21701-5011.

SO INFECT IMMUN, (1988) 56 (7), 1807-1813.
CODEN: INFIBR. ISSN: 0019-9567.
FS BA; OLD
LA English

L16 ANSWER 121 OF 123 MEDLINE
AN 87208610 MEDLINE
DN 87208610 PubMed ID: 3107286
TI [Quantitative evaluation of a population of immunocompetent cells having a **receptor** for the **anthrax protective antigen**].
Kolichestvennaia otsenka populiatsii immunokompetentnykh kletok, imeiushchikh retseptor k protektivnomu sibireiazvennomu antigenu.
AU Meretskov V V; Lebedinskii V A; Garin N S; Smirnov V S
SO ZHURNAL MIKROBIOLOGII, EPIDEMIOLOGII I IMMUNOBIOLOGII, (1987 Feb) (2) 54-7.
Journal code: 0415217. ISSN: 0372-9311.
CY USSR
DT Journal; Article; (JOURNAL ARTICLE)
LA Russian
FS Priority Journals
EM 198705
ED Entered STN: 19900303
Last Updated on STN: 19900303
Entered Medline: 19870522

L16 ANSWER 122 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1987:220396 BIOSIS
DN BR32:106270
TI PURIFICATION OF **ANTHRAX** TOXIN **PROTECTIVE ANTIGEN** COMPONENT AND CHARACTERIZATION OF ITS BINDING INTERACTION WITH BOVINE KIDNEY CELLS.
AU MARTIN D D; RESNICK I G
CS UTAH STATE UNIV., LOGAN, UTAH.
SO 87TH ANNUAL MEETING OF THE AMERICAN SOCIETY FOR MICROBIOLOGY, ATLANTA, GEORGIA, USA, MARCH 1-6, 1987. ABSTR ANNU MEET AM SOC MICROBIOL. (1987) 87 (0), 29.
CODEN: ASMACK. ISSN: 0094-8519.
DT Conference
FS BR; OLD
LA English

L16 ANSWER 123 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 53
AN 1983:161494 BIOSIS
DN BA75:11494
TI **ANTHRAX** BACILLUS-**ANTHRACIS** TOXIN EDEMA FACTOR A BACTERIAL ADENYLATE CYCLASE THAT INCREASES CYCLIC AMP CONCENTRATIONS IN EUKARYOTIC CELLS.
AU LEPPLA S H
CS DEP. APPLIED TOXIN RES., PATHOL. DIV., U.S. ARMY MED. RES. INST. INFECTIOUS DISEASES, FORT DETRICK, FREDERICK, MARYLAND 21701.
SO PROC NATL ACAD SCI U S A, (1982) 79 (10), 3162-3166.
CODEN: PNASA6. ISSN: 0027-8424.
FS BA; OLD
LA English

=> d clm 11

L16 ANSWER 11 OF 123 USPATFULL
CLM What is claimed is:

1. A system for sensing at least one analyte in a sample comprising: a sensor element having a **receptor** site; and a host molecule, wherein the host molecule interacts with the **receptor** site of the sensor element and the analyte as an adapter between the analyte and the **receptor** site so that the sensor element directly produces a detectable signal.

2. A system for sensing a plurality of different analytes comprising: at least one sensor element, each sensor element comprising a pore and having a **receptor** site; and a plurality of different host molecules, wherein the host molecules each interact with a **receptor** site of a sensor element and at least one of the different analytes as an adapter between the analyte and the **receptor** site so that the sensor element directly produces a detectable signal.

3. A biosensor for detecting an analyte in a sample comprising: a bilayer separating the biosensor into a first compartment and a second compartment; a sensor element disposed in the bilayer so that it forms a channel in the bilayer; and a host molecule, wherein the host molecule interacts with a **receptor** site on the sensor element and the analyte as an adapter between the analyte and the **receptor** site so that the sensor element directly produces a detectable signal.

4. A system for sensing at least one analyte in a sample comprising: a sensor element having a **receptor** site; and a host molecule, wherein the host molecule interacts with the **receptor** site of the sensor element and the analyte as a carrier to deliver the analyte to the **receptor** site so that the sensor element directly produces a detectable signal.

5. A system for sensing a plurality of different analytes comprising: a plurality of different sensor elements, each sensor element comprising a pore and having a **receptor** site; and a plurality of different host molecules, wherein the host molecules each interact with a **receptor** site of one of the plurality of different sensor elements and one of the different analytes as a carrier to deliver the analyte to the **receptor** site so that the sensor element directly produces a detectable signal.

6. A biosensor for detecting an analyte in a sample comprising: a bilayer separating the biosensor into a first compartment and a second compartment; a sensor element disposed in the bilayer so that it forms a channel in the bilayer; and a host molecule, wherein the host molecule interacts with a **receptor** site on the sensor element and the analyte as a carrier to deliver the analyte to the **receptor** site so that the sensor element directly produces a detectable signal.

7. The system of any one of claim 1 or 4 wherein sensing comprises stochastic sensing.

8. The system of claim 1 wherein the host molecule is non-covalently attached to the **receptor** site.

9. The system of claim 1 wherein the host molecule is covalently attached to the **receptor** site.

10. The system of any one of claim 1 or 4 wherein the system further comprises a bilayer and the sensor element comprises a channel disposed in the bilayer.

11. The system of any one of claim 1 or 4 wherein the system further

comprises a bilayer apparatus, the bilayer apparatus comprising a bilayer separating the bilayer apparatus into a first compartment and a second compartment and wherein the sensor element is disposed in the bilayer so that it forms a channel in the bilayer.

12. The system of claim 11 wherein the sensor element is disposed in the first compartment so that it forms a channel in the bilayer and the host molecule is disposed in the second compartment.

13. The system of claim 11 wherein the sensor element is disposed in the first compartment so that it forms a channel in the bilayer and the host molecule is disposed in the first compartment, the second compartment or both compartments.

14. The system of any one of claim 1 or 4 wherein sensing comprises identifying the analyte.

15. The system of any one of claim 1 or 4 wherein sensing comprises quantitating the analyte.

16. The system of any one of claim 1 or 4 wherein the host molecule is selected from the group consisting of a cyclodextrin, a poly(ethylene glycol) molecule, a synthetic polymer, an oligonucleotide, an aptamer, a peptide polymer and an oligosaccharide.

17. The system of claim 1 wherein the host molecule is a cyclodextrin.

18. The system of claim 17 wherein the cyclodextrin is .beta.-cyclodextrin (.beta.CD).

19. The system of claim 17 wherein the cyclodextrin is s.sub.7.beta.CD.

20. The system of any one of claim 1 or 4 wherein the sensor element is a protein.

21. The system of claim 20 wherein the protein is selected from the group consisting of a transmembrane pore, an enzyme, an antibody and a **receptor**.

22. The system of any one of claim 1 or 4 wherein the sensor element comprises a pore.

23. The system of claim 22 wherein the sensor element comprises a genetically engineered transmembrane protein pore.

24. The system of claim 22 wherein the sensor element is an .alpha.-Hemolysin (.alpha.HL) pore.

25. The system of claim 24 wherein the sensor element is a wild-type .alpha.-Hemolysin (.alpha.HL) pore.

26. The system of claim 24 wherein the sensor element is a genetically engineered or mutant .alpha.-Hemolysin (.alpha.HL) pore.

27. The system of any one of claim 1 or 4 wherein the system senses at least two analytes.

28. The system of any one of claim 1 or 4 wherein the signal comprises a change in electrical current.

29. The system of any one of claim 1 or 4 wherein the signal comprises a change in the magnitude and duration of the change in the current.

30. The system of any one of claim 1 or 4 wherein the analyte is an organic molecule.

31. The system of any one of claim 1 or 4 wherein the analyte is not charged.

32. The system of any one of claim 1 or 4 wherein the signal is selected from the group consisting of a change in fluorescence, a change in electrical current and a change in force.

33. The biosensor of any one of claim 3 or 6 wherein the sensor element is disposed in the first compartment so that it forms a channel in the bilayer and the host molecule is disposed in the second compartment.

34. The biosensor of claim 33 wherein the host molecule is disposed in the second compartment substantially simultaneously with the addition of the sample to the second compartment.

35. The biosensor of any one of claim 3 or 6 wherein the sensor element is disposed in the first compartment so that it forms a channel in the bilayer and the host molecule is disposed in the first compartment.

36. The biosensor of claim 35 wherein the host molecule is disposed in the first compartment substantially simultaneously with the addition of the sample to the first compartment.

37. The system of one of claim 2 or 5, wherein the system comprises a plurality of different sensor elements.

38. The system of claim 2, wherein one of more of the host molecules is capable of interacting with one or more of the different analytes as an adapter between the analyte and the **receptor** site and each interacts with the **receptor** site of a sensor element and one analyte molecule at a given time.

39. The system of claim 5, wherein one of more of the host molecules is capable of interacting with one or more of the different analytes as a carrier to deliver the analyte to the **receptor** site and each interacts with the **receptor** site of a sensor element and one analyte molecule at a given time.

=> d ab 113

L16 ANSWER 113 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

=> d his

(FILE 'HOME' ENTERED AT 16:14:58 ON 29 AUG 2002)

FILE 'BIOSIS, MEDLINE, AGRICOLA, EMBASE, CABA, WPIDS, JAPIO, BIOTECHDS, LIFESCI, CAPLUS, USPATFULL, USPAT2' ENTERED AT 16:15:12 ON 29 AUG 2002

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                E COLLIER R JOHN/AU
L1          293 S E1-E3
                E BRADLEY KENNETH A/AU
L2           11 S E2-E3
                E BRADLEY K A/AU
L3          257 S E2-E3
                E MOGRIDGE JEREMY/AU
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L4            30 S E3
                 E MORGRIDGE J/AU
                 E MOGRIDGE J/AU
L5            50 S E3
                 E YOUNG JOHNA T/AU
                 E YOUNG JOHN A T/AU
L6            76 S E3
                 E YOUNGJ A T/AU
                 E YOUNG J A T/AU
L7            99 S E3-E4
L8           785 S L1-L7
L9           117 S L8 AND ANTHRA?
L10           92 S L9 AND PROTECTIVE ANTIGEN
L11           44 DUP REM L10 (48 DUPLICATES REMOVED)
L12           13 S L11 AND RECEPTOR
L13       268052 S ANTHRA?
L14         1553 S L13 AND PROTECTIVE (5A) ANTIGEN
L15          314 S L14 AND RECEPTOR
L16          123 DUP REM L15 (191 DUPLICATES REMOVED)

=> s l16 and (nucleic acid or DNA or cDNA or polynucleotide)
   8 FILES SEARCHED...
  10 FILES SEARCHED...
  11 FILES SEARCHED...
L17           33 L16 AND (NUCLEIC ACID OR DNA OR CDNA OR POLYNUCLEOTIDE)

=> d bib ab 1-33

L17  ANSWER 1 OF 33  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN   1999:263318  BIOSIS
DN   PREV199900263318
TI   Endoprotease PACE4 is Ca2+-dependent and temperature-sensitive and can
     partly rescue the phenotype of a furin-deficient cell strain.
AU   Sucic, Joseph F. (1); Moehring, Joan M.; Inocencio, Noel M.; Luchini,
     Jason W.; Moehring, Thomas J.
CS   (1) Biology Department, University of Michigan-Flint, 303 East Kearsley
     St., Flint, MI, 48502-1950 USA
SO   Biochemical Journal, (May 1, 1999) Vol. 339, No. 3, pp. 639-647.
     ISSN: 0264-6021.
DT   Article
LA   English
SL   English
AB   PACE4 is a member of the eukaryotic subtilisin-like endoprotease family.
     The expression of human PACE4 in RPE.40 cells (furinnull mutants derived
     from Chinese hamster ovary K1 cells) resulted in the rescue of a number of
     wild-type characteristics, including sensitivity to Sindbis virus and the
     ability to process the low-density-lipoprotein receptor-related
     protein. Expression of PACE4 in these cells failed to restore wild-type
     sensitivity to Pseudomonas exotoxin A. Co-expression of human PACE4 in
     these cells with either a secreted form of the human insulin pro-
     receptor or the precursor form of von Willebrand factor resulted
     in both proproteins being processed; RPE.40 cells were unable to process
     either precursor protein in the absence of co-expressed PACE4. Northern
     analysis demonstrated that untransfected RPE.40 cells express mRNA species
     for four PACE4 isoforms, suggesting that any endogenous PACE4 proteins
     produced by these cells are either non-functional or sequestered in a
     compartment outside of the secretory pathway. In experiments in vitro,
     PACE4 processed diphtheria toxin and anthrax toxin
     protective antigen, but not Pseudomonas exotoxin A. The
     activity of PACE4 in vitro was Ca2+-dependent and, unlike furin, was
     sensitive to temperature changes between 22 and 37 degreeC. RPE.40 cells
     stably expressing human PACE4 secreted an endoprotease with the same Ca2+
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dependence and temperature sensitivity as that observed in membrane fractions of these cells assayed in vitro. These results, in conjunction with other published work, demonstrate that PACE4 is an endoprotease with more stringent substrate specificity and more limited operating parameters than furin.

L17 ANSWER 2 OF 33 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 93353907 EMBASE
DN 1993353907
TI Characterization of Clostridium perfringens iota-toxin genes and expression in Escherichia coli.
AU Perelle S.; Gibert M.; Boquet P.; Popoff M.R.
CS Laboratoire des Toxines Microbiennes, Institut Pasteur, 28 Rue du Dr. Roux,75724 Paris Cedex 15, France
SO Infection and Immunity, (1993) 61/12 (5147-5156).
ISSN: 0019-9567 CODEN: INFIBR
CY United States
DT Journal; Article
FS 004 Microbiology
LA English
SL English
AB The iota toxin which is produced by Clostridium perfringens type E, is a binary toxin consisting of two independent polypeptides: Ia, which is an ADP- ribosyltransferase, and Ib, which is involved in the binding and internalization of the toxin into the cell. Two degenerate oligonucleotide probes deduced from partial amino acid sequence of each component of C. spiroforme toxin, which is closely related to the iota toxin, were used to clone three overlapping **DNA** fragments containing the iota-toxin genes from C. perfringens type E plasmid **DNA**. Two genes, in the same orientation, coding for Ia (387 amino acids) and Ib (875 amino acids) and separated by 243 noncoding nucleotides were identified. A predicted signal peptide was found for each component, and the secreted Ib displays two domains, the propeptide (172 amino acids) and the mature protein (664 amino acids). The Ia gene has been expressed in Escherichia coli and C. perfringens, under the control of its own promoter. The recombinant polypeptide obtained was recognized by Ia antibodies and ADP-ribosylated actin. The expression of the Ib gene was obtained in E. coli harboring a recombinant plasmid encompassing the putative promoter upstream of the Ia gene and the Ia and Ib genes. Two residues which have been found to be involved in the NAD+ binding site of diphtheria and pseudomonas toxins are conserved in the predicted Ia sequence (Glu-14 and Trp-19). The predicted amino acid Ib sequence shows 33.9% identity with and 54.4% similarity to the **protective antigen** of the **anthrax** toxin complex. In particular, the central region of Ib, which contains a predicted transmembrane segment (Leu-292 to Ser-308), presents 45% identity with the corresponding **protective antigen** sequence which is involved in the translocation of the toxin across the cell membrane.

L17 ANSWER 3 OF 33 WPIDS (C) 2002 THOMSON DERWENT
AN 2001-218343 [22] WPIDS
DNC C2001-065177
TI Novel fusion protein for modifying apoptosis in target cell and reducing apoptosis after transient ischemic neuronal injury, has two domains which targets protein to a cell and modifies apoptotic response of cell.
DC B04 D16
IN COLLIER, R J; LIU, X; YOULE, R J
PA (HARD) HARVARD COLLEGE; (USSH) US DEPT HEALTH & HUMAN SERVICES
CYC 94
PI WO 2001012661 A2 20010222 (200122)* EN 55p
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM
DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC
LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE
SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

AU 2000069061 A 20010313 (200134)

ADT WO 2001012661 A2 WO 2000-US22293 20000815; AU 2000069061 A AU 2000-69061 20000815

FDT AU 2000069061 A Based on WO 200112661

PRAI US 1999-149220P 19990816

AB WO 200112661 A UPAB: 20010421

NOVELTY - A functional apoptosis-modifying fusion protein (I) capable of binding a target cell and integrating into or crossing a cellular membrane of the target cell, comprising at least two domains, one of which targets the fusion protein to the target cell and another of which modifies an apoptotic response of the target cell, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated **nucleic acid** molecule (II) encoding (I);

(2) a recombinant **nucleic acid** molecule (III) comprising a promoter sequence operably linked to (II);

(3) a transgenic cell comprising (III);

(4) preparation of (I);

(5) a composition (IV) comprising (I), its analog or mimetic;

(6) a pharmaceutical composition comprising (IV);

(7) a combined pharmaceutical composition comprising (I) and **anthrax protective antigen** (PA) to enable measurable transport of (I) into a target cell; and

(8) a protein analog, derivative or mimetic of (I).

ACTIVITY - Nootropic; Neuroprotective; Cytostatic; Cerebroprotective; Anticonvulsant.

MECHANISM OF ACTION - Modulator of apoptosis.

The apoptosis inhibiting effect of BCL-xL-diphtheria toxin **receptor** binding domain (DTR) was studied. The apoptosis inhibition activity of zVAD-fmk and Boc-D-fmk, potent caspase inhibitors was compared with that of BCL-xL-DTR. HeLa cells were plated at a density of 1 multiply 105 cells/well, infected with poliovirus at an multiplicity of infection (MOI) of 1 plaque forming units (pfu)/cell and immediately treated with negative control peptide zFA-fmk at 20 micro M, BCL-xL-DTR at 0.48 micro M or peptides zVAD-fmk or Boc-D-fmk at 20 micro M. Cell viability was assessed. BCL-xL-DTR at 0.48 micro M blocked cell death to a greater extent than either zVAD-fmk or Boc-D-fmk at 20 micro M, indicating a strong inhibition of apoptosis pathway by BCL-xL-DTR.

USE - (I) is useful for modifying (inhibiting or enhancing) apoptosis in a target cell, such as neuron, lymphocyte, cancer, neoplasm, macrophage, epithelial, stem, tumor or hyper-proliferative cell or an adipocyte. (I) is also useful for reducing apoptosis in a subject after transient ischemic neuronal injury, especially spinal cord injury (claimed). (I) may be used to treat various diseases and injury conditions through inhibition or enhancement of apoptotic cellular response, including neurodegenerative disorders such as Alzheimer's disease, Huntington's disease, spinal muscular atrophy, stroke episodes and unregulated cell growth as in tumors and various cancers.

ADVANTAGE - Apoptosis-modifying fusion proteins can be delivered effectively throughout the body and targeted to selective tissue and cells.

Dwg.0/12

L17 ANSWER 4 OF 33 BIOTECHDS COPYRIGHT 2002 THOMSON DERWENT AND ISI

AN 2002-06073 BIOTECHDS

TI Screening Bacillus **anthracis** toxicity inhibitor (T) by generating recombinant **protective antigen** 32,

comparing fluorescence of cells contacted with PA32-fluorescent marker fusion protein before, after contact with T;
vector-mediated **protective antigen**-32 and enhanced green fluorescent protein reporter gene transfer, expression in human A549 cell, single chain antibody and **nucleic acid** vaccine for recombinant protein production, drugscreening and bacterium infection therapy and gene therapy

AU CIRINO N M; JACKSON P J; LEHNERT B E
PA UNIV CALIFORNIA
PI US 6329156 11 Dec 2001
AI US 1999-273839 22 Mar 1999
PRAI US 1999-273839 22 Mar 1999
DT Patent
LA English
OS WPI: 2002-121130 [16]
AB DERWENT ABSTRACT: NOVELTY - A recombinant **protective antigen** (PA)32 **DNA** fragment from PA83 of Bacillus **anthracis** (Ba) is generated, fused to enhanced green fluorescent protein (EGFP) and expressed. Resulting EGFP-PA32 protein is mixed with Ba toxicity inhibitor (T) and contacted with mammalian cell sample (CS) to form fluorescent CS, and fluorescence (F) of the cells is compared with (F) of cells not contacted with (T). DETAILED DESCRIPTION - Screening inhibitors of the toxicity of Ba involves: (a) generating the recombinant PA32 **DNA** fragment which has a fully defined sequence of 867 nucleotides (S7) as given in specification, from region 4 of PA83 of Ba and ligating the PA32 **DNA** fragment to EGFP, to form EGFP-PA32; (b) expressing the EGFP-PA32 produce the EGFP-PA32 protein; (c) contacting the EGFP-PA32 protein with individual cells in a first sample of mammalian cells, thereby generating a first sample of fluorescent cells; (d) measuring (F) from individual cells in the first sample of fluorescent cells; (e) mixing EGFP-PA32 protein with a potential toxicity inhibitor of Ba; (f) contacting the mixture of the EGFP-PA32 protein and the potential toxicity (T) with individual cells in a second sample of mammalian cells, forming thereby a second sample of fluorescent cells; (g) measuring (F) from individual cells in the second sample of fluorescent cells; and (h) comparing (F) from individual cells in the first sample of fluorescent cells with (F) from individual cells in the second sample of (F) cells, whereby the effectiveness of the toxicity (T) was determined from the decrease of (F) from individual cells from the second sample of fluorescent cells relative to (F) from individual cells in the first sample of fluorescent cells. WIDER DISCLOSURE - The following are disclosed: (1) generating recombinant PA fragment containing domain 4 of PA83 to compete with native PA83 for its receptors, thereby inhibiting the first step required for toxin complex formation; and (2) inhibiting the toxicity of Ba by: (a) introducing the recombinant fragment PA32 protein into an exposed individual, where PA83 is competitively inhibited from binding to the cells of the exposed individual; (b) introducing human scFv4 antibody into an exposed individual, whereby the scFv4 binds to PA83, thereby preventing PA83 from binding to the cells of the exposed individuals; (c) introducing the recombinant fragment PA32 protein into an individual, whereby antibodies suitable for preventing PA83 from binding to the cells of the individual exposed to Ba are generated by the individual, that is immunization occurs; (d) introducing **DNA**-encoding PA32 into the genetic material of host cells, whereby the host cell machinery transcribes and translates PA32 which secretes the recombinant, synthetic antibody fragment, thereby acting as a **DNA** vaccine; or (e) introducing **DNA**-encoding scFv into the genetic material of host cells, whereby the host cell machinery transcribes and translates scFv, which secretes the recombinant, synthetic antibody fragment. BIOTECHNOLOGY - Preferred Method: The mammalian cells are A549 human bronchial epithelial cells. (F) from individual cells of first and second sample is measured

using flow cytometry. ACTIVITY - Antibacterial. No supporting data is given. MECHANISM OF ACTION - Bacillus **anthracis** toxicity inhibitor. USE - The method is useful for screening (T) of toxicity of Ba (claimed). PA32 may be used to inhibit the toxicity of Ba. ADMINISTRATION - No specific administration details are given. ADVANTAGE - The method can be used as a rapid assay for small molecule (T) of PA binding to cell receptors. EXAMPLE - **Protective antigen** (PA)83 was purified as described in purification of **anthrax**-toxin components by high-performance anion-exchange; gel-filtration and hydrophobic-interaction chromatography by C. P. Quinn et al., J. Biochem. 252, 753 (1988). Clarified supernatant was collected from a 20 L culture of pXO2 cured Sterne strain Bacillus **anthracis**. A 20% ammonium sulfate precipitation was used to enrich PA83 relative to other secreted proteins. Subsequent fast protein liquid chromatography (FPLC) purifications were performed using MONO-Q (RTM) and gel filtration (SEPHADEX G-75 (RTM)) columns. The final protein preparation was greater than 90% pure. Purification of recombinant **anthrax** proteins was performed by immobilized metal affinity chromatography (IMAC) in a single step. All IMAC purified proteins were greater than 95% homogeneous after elution as determined by SDS-polyacrylamide gel electrophoresis. A recombinant PA comprised of the carboxy-terminal 32 kDa was highly soluble in Escherichia coli and did not appear to be toxic to the bacteria. PA32 was cloned as a fusion protein with a enhanced green fluorescent protein (EGFP) attached to its amino terminus. The EGFP-PA32 fusion was designed for use in flow cytometry assay where inhibitors of PA **receptor** binding could be analyzed. Chimeric EGFP-EF32 were expressed and purified. Synthetic, recombinant, single-chain Fv from a naive phage display library were biopanned against PA83. Following 3 rounds of selection, 60 of 90 isolates showed PA binding ability, as determined by enzyme linked immunosorbent assay (ELISA). Fingerprint analysis revealed 7 unique isolates, of which 5 (scFv1, scFv4, scFv5, scFv12, scFv24) with the highest ELISA scores were chosen for further analysis. These scFv were expressed and purified to isolate monomeric scFv. scFv5, showed greater than 90% multimerization and was therefore excluded from subsequent analysis. This procedure yielded greater than 95% pure antibodies. PA83 was coupled to a BIAcore CM5 chip and four dilutions of each of the purified, monomeric scFv were used to determine equilibrium dissociation constants (Kd). All scFv tested showed similar affinities. These scFv were further assessed for their ability to recognize the recombinant PA32 fragment. PA83, EGFP-PA32, PA32 and EGFP-EF32 were coupled to different channels on a single BIAcore CM5 flowcell. Different scFv were sequentially passed over each channel of the chip and their affinity determined. All ligands were coupled at 1000 RU and a single concentration of analyte was assessed. Two scFvs (1 and 4) showed similar affinities for PA83 and PA32 ligands while scFv12 showed only non-specific binding to PA32 proteins. These data indicated that the targets for scFv1 and scFv4 lie within domains 3 or 4 of PA while the antigenic site for scFv12 was outside this region. Further experiments carried out showed that PA32 fragment was recognized similar to natural PA83 and internalized into cytoplasmic vesicles. A flow cytometric assay developed using the EGFP-PA32 fusion protein. Human A549 cells were used as target cells because of their low autofluorescence and minimal phagocytic activity. EGFP alone or the EGFP-EF32 fusion was used to evaluate nonspecific binding by these cells. A 4-fold enhanced signal was observed from specific EGFP-PA32 bound to cells compared to non-specific EGFP binding alone. To confirm that EGFP-PA32 was binding to the PA specific **receptor**, competition with different concentrations of natural PA83 or unlabeled PA32 was assessed. There was a statistically significant (p less than 0.0001) linear inhibition of fluorescent-PA32 binding by unlabeled PA molecules. For a 1:1 stoichiometry of PA/**receptor** binding, a 50% inhibition by an equimolar concentration of unlabeled PA would be expected (i.e., 50%

EGFP-PA32, 50% competitor). This data confirmed specificity and indicated little or no cooperativity in PA/**receptor** interactions. Flow cytometric analysis was subsequently used to screen scFv for their ability to disrupt PA-**receptor** interactions. Incubation of scFv4 with EGFP-PA32 at a 1:1 molar ratio was able to significantly (greater than 80%) abolish **receptor**-mediated binding of EGFP-PA32 to A549 cells. The scFv1, which can recognize EGFP-PA32 showed minimal inhibition of EGFP-PA32 binding by this assay. This indicated that it did not recognize or mask an essential structure necessary for **receptor** recognition. These data indicated the flow cytometric assay was a sensitive and specific method to identify molecules which inhibit **receptor**-mediated **anthrax** toxin binding, and that one of the scFv selected has the potential to inhibit PA binding to cells in a therapeutically useful fashion.(14 pages)

L17 ANSWER 5 OF 33 BIOTECHDS COPYRIGHT 2002 THOMSON DERWENT AND ISI
AN 2001-07648 BIOTECHDS
TI Novel fusion protein for modifying apoptosis in target cell and reducing apoptosis after transient ischemic neuronal injury, has 2 domains which targets protein to a cell and modifies apoptotic response of cell; plasmid pcDNA3-mediated diphtheria toxin **receptor** binding domain and BCL-xl domain gene transfer and expression in Escherichia coli
AU Youle R J; Liu X; Collier R J
PA U.S.Dep.Health-Hum.Serv.; Nat.Inst.Health-Rockville; Univ.Harvard
LO Rockville, MD, USA; Cambridge, MA, USA.
PI WO 2001012661 22 Feb 2001
AI WO 2000-US22293 15 Aug 2000
PRAI US 1999-149220 16 Aug 1999
DT Patent
LA English
OS WPI: 2001-218343 [22]
AB A functional apoptosis-modifying fusion protein (411, 485 or 567 amino acids) capable of binding a target cell and integrating into or crossing a cellular membrane of the target cell, containing at least 2 domains, one of which targets the fusion protein to the target cell (e.g. diphtheria toxin **receptor** binding domain) and another of which modifies an apoptotic response of the target cell (e.g. BCL-xl), is new. Also claimed are: a **nucleic acid** (1,236, 1,704 or 1,455 bp) encoding the protein; a recombinant **nucleic acid** containing a promoter sequence linked to the **nucleic acid**; a transgenic cell containing the **nucleic acid**; preparation of the fusion protein; a composition containing the protein; a pharmaceutical composition containing the composition; a combined pharmaceutical composition containing the protein and **anthrax protective antigen** to enable measurable transport of the protein into a target cell; and a protein analog, derivative or mimetic of the protein. The protein is useful for modifying apoptosis in a target cell, such as neuron, lymphocyte, cancer etc. In an example, plasmid pcDNA3 was used to transform Escherichia coli BL21 (DE3). (55pp)

L17 ANSWER 6 OF 33 CAPLUS COPYRIGHT 2002 ACS
AN 2002:449716 CAPLUS
DN 137:29035
TI Sequences of a human **receptor** for B. **anthracis** toxin and therapeutical uses
IN Young, John A. T.; Bradley, Kenneth A.; Collier, Robert J.; Mogridge, Jeremy S.
PA Wisconsin Alumni Research Foundation, USA
SO PCT Int. Appl., 45 pp.
CODEN: PIXXD2

DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002046228	A2	20020613	WO 2001-US30941	20011003

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2000-251481P P 20001205

AB The present invention discloses sequences of a human **receptor** for B. **anthracis** toxin and its therapeutical uses. Specifically, the present invention relates to a human **anthrax** toxin **receptor** and polynucleotides encoding the **receptor** as well as related proteins and polynucleotides, vectors contg. the polynucleotides and proteins, host cells contg. related **polynucleotide** mols., and cells displaying no **anthrax** toxin **receptor** on an exterior surface of the cells. The present invention also relates to methods for identifying mols. that bind the **anthrax** toxin **receptor** and mols. that reduce the toxicity of **anthrax** toxin. Finally, the present invention provides methods for treating human and non-human animals suffering from **anthrax**.

L17 ANSWER 7 OF 33 CAPLUS COPYRIGHT 2002 ACS
AN 1999:27954 CAPLUS
DN 130:77075
TI Targetting and uptake of **DNA** by animal cells by **receptor** -mediated endocytosis using fusion protein of toxins and **DNA** -binding proteins
IN Grandi, Guido
PA Chiron S.P.A., Italy
SO PCT Int. Appl., 85 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9859065	A1	19981230	WO 1998-IB1005	19980618

W: JP, US

RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE

PRAI GB 1997-13122 19970620

AB A method of using **receptor**-mediated endocytosis to increase the efficiency of **DNA** uptake by eukaryotic cells is described. The method uses fusion proteins of **receptor**-binding domains of toxins, therefore lacking the domains necessary for toxic activity, and **DNA**-binding domains. These fusion proteins are taken up by the **receptor** for the toxin and the **DNA** it is bound to is incorporated into the endosome. When the endosome is internalized, the complex is released and the protein stripped from the **DNA** leaving it free to become part of the host cell genome. A fusion protein of the heat-labile enterotoxin of Escherichia coli and the histone H1-like protein of Bordetella pertussis was prepd. by expression of the cloned gene. The protein was shown to retain **DNA** binding activity.

Similarly, a fusion protein of diphtheria toxin and GAL4 was shown to have **DNA** binding and to retain the normal binding of the toxin to Vero cells. The fusion protein was also rapidly internalized by Vero cells.

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 8 OF 33 USPATFULL
AN 2002:201863 USPATFULL
TI Dendritic cell **receptor**
IN Hart, Derek N., Christchurch, NEW ZEALAND
PA The Corporation of the Trustees of the Sisters of Mercy in Queensland, Queensland, AUSTRALIA (non-U.S. corporation)
PI US 6432666 B1 20020813
WO 9745449 19971204
AI US 1999-194612 19990318 (9)
WO 1997-NZ68 19970529
19990318 PCT 371 date
PRAI NZ 1996-286692 19960529
DT Utility
FS GRANTED
EXNAM Primary Examiner: Eyler, Yvonne; Assistant Examiner: Hamud, Fozia
LREP Nixon & Vanderhye
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN 19 Drawing Figure(s); 15 Drawing Page(s)
LN.CNT 1781
AB An isolated human dendritic cell **receptor** comprising amino acid sequences selected from: TVDCNDNQPGAICYYSGNETEKEVKPVDSVKCPSPVLNTPWI PFQNCCYN FIITKNRHMATTQDEVQSTCEKLHPKSHILSIRDEKENNFVLEQLLYFNYMA SWVMLGITYRNNSL amino acid at position 1208-1323 of SEQ ID NO:1 and SQHRLFHLHSQKCLGLDITKSVNELRMFSCDSSAML amino acid at position 71-106 of SEQ ID NO:1.

L17 ANSWER 9 OF 33 USPATFULL
AN 2002:188260 USPATFULL
TI Analyte sensing mediated by adapter/carrier molecules
IN Bayley, Hagan, College Station, TX, United States
Braha, Orit, College Station, TX, United States
Gu, LiQun, Bryan, TX, United States
PA The Texas A&M University System, College Station, TX, United States (U.S. corporation)
PI US 6426231 B1 20020730
AI US 1999-441376 19991117 (9)
PRAI US 1998-109034P 19981118 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Chin, Christopher L.
LREP Baker Botts L.L.P.
CLMN Number of Claims: 39
ECL Exemplary Claim: 1
DRWN 33 Drawing Figure(s); 9 Drawing Page(s)
LN.CNT 1747
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention relates to an improved method and system for sensing of one or more analytes. A host molecule, which serves as an adapter/carrier, is used to facilitate interaction between the analyte and the sensor element. A detectable signal is produced reflecting the identity and concentration of analyte present.

L17 ANSWER 10 OF 33 USPATFULL
AN 2002:172486 USPATFULL
TI Dendritic cell co-stimulatory molecules

IN Pardoll, Drew M., Brookville, MD, UNITED STATES
Tsuchiya, Haruo, Baltimore, MD, UNITED STATES
Gorski, Kevin S., Baltimore, MD, UNITED STATES
Tseng, Su-Yi, Baltimore, MD, UNITED STATES
PI US 2002091246 A1 20020711
AI US 2001-794210 A1 20010228 (9)
PRAI US 2000-200580P 20000428 (60)
US 2000-240169P 20001013 (60)
DT Utility
FS APPLICATION
LREP VENABLE, Post Office Box 34385, Washington, DC, 20043-9998
CLMN Number of Claims: 120
ECL Exemplary Claim: 1
DRWN 8 Drawing Page(s)
LN.CNT 3534
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A novel costimulatory protein molecule, B7-DC, which is a member of the B7 family, is described as is **DNA** coding therefor and expression vectors comprising this **DNA**. B7-DC protein, fragments, fusion polypeptides/proteins and other functional derivatives, and transformed cells expressing B7-DC are useful in vaccine compositions and methods. Compositions and methods are disclosed for inducing potent T cell mediated responses that can be harnessed for anti-tumor and anti-viral immunity.

L17 ANSWER 11 OF 33 USPATFULL
AN 2002:136555 USPATFULL
TI Methods of modulating an immune response to antigen, and cells for use in the method
IN Segal, Andrew H., Boston, MA, United States
PA Whitehead Institute for Biomedical Research, Cambridge, MA, United States (U.S. corporation)
PI US 6403080 B1 20020611
AI US 1999-339523 19990624 (9)
RLI Division of Ser. No. US 1997-826259, filed on 27 Mar 1997, now patented, Pat. No. US 5951976
PRAI US 1996-14364P 19960328 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Bansal, Geetha P.
LREP Williams, Kathleen Madden, Palmer & Dodge, LLP
CLMN Number of Claims: 25
ECL Exemplary Claim: 1
DRWN 0 Drawing Figure(s); 0 Drawing Page(s)
LN.CNT 2153
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are methods and compositions wherein opsonin-enhanced cells, that is, cells which have been 1) modified so as to express an opsonin from a recombinant **nucleic acid**, 2) modified so as to express higher levels of an endogenous opsonin, or 3) mixed with an exogenous opsonin, when administered to a subject, modulate the immune response in the recipient to a selected antigen or antigens contained in or attached to the cells.

L17 ANSWER 12 OF 33 USPATFULL
AN 2002:105667 USPATFULL
TI Inhibition of mitogen-activated protein kinase (MAPK) pathway: a selective therapeutic strategy against melanoma
IN Koo, Han-Mo, Kentwood, MI, UNITED STATES
Vande Woude, George F., Ada, MI, UNITED STATES
PI US 2002054869 A1 20020509
AI US 2001-942940 A1 20010831 (9)

PRAI US 2000-229290P 20000901 (60)
US 2001-285690P 20010424 (60)
DT Utility
FS APPLICATION
LREP VENABLE, BAETJER, HOWARD AND CIVILETTI, LLP, P.O. BOX 34385, WASHINGTON, DC, 20043-9998
CLMN Number of Claims: 21
ECL Exemplary Claim: 1
DRWN 16 Drawing Page(s)
LN.CNT 2335
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Inhibitors of the MAPK pathway, including MEK-directed proteases and small molecule inhibitors, are cytotoxic to human melanoma cells in vitro and in vivo via apoptotic mechanisms. These compounds are used to kill melanoma cells and to treat subjects with melanoma, either alone or in combination with other therapeutic modalities.

L17 ANSWER 13 OF 33 USPATFULL
AN 2002:98896 USPATFULL
TI Methods for protection against lethal infection with bacillus **anthracis**
IN Galloway, Darrel R., Dublin, OH, UNITED STATES
Mateczun, Alfred J., Albuquerque, NM, UNITED STATES
PI US 2002051791 A1 20020502
AI US 2000-747521 A1 20001221 (9)
PRAI US 1999-171459P 19991222 (60)
DT Utility
FS APPLICATION
LREP NAVAL MEDICAL RESEARCH CENTER, ATTN: (CODE 00L), 503 ROBERT GRANT AVENUE, SILVER SPRING, MD, 20910-7500
CLMN Number of Claims: 30
ECL Exemplary Claim: 1
DRWN 8 Drawing Page(s)
LN.CNT 1459
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods of intducing an immune response which protects a susceptible animal subject from lethal infection with Bacillus **anthracis** (B. **anthracis**) are provided. One method comprises administering B. **anthracis** lethal factor (LF) or an immunogenic fragment thereof to the subject. A second method comprises administering LF or an immunogenic fragment thereof and the B **anthracis protective antigen** (PA) to the subject. A third method comprises administering a **polynucleotide** which encodes B. **anthracis** LF or an immunogenic fragment thereof to the subject. A fourth method comprises administering a **polynucleotide** which encodes LF or an immunogenic fragment thereof and a **polynucleotide** which encodes the B. **anthracis** PA to the subject. The present invention also relates to a protein or peptide based-immunogenic composition for preparing a vaccine which is capable of prophylactically protecting a subject against lethal effects of infection with B. **anthracis**.

L17 ANSWER 14 OF 33 USPATFULL
AN 2002:92073 USPATFULL
TI Targeting antigens to the MHC class I processing pathway with an **anthrax** toxin fusion protein
IN Klimpel, Kurt, Gaithersburg, MD, UNITED STATES
Goletz, Theresa J., Kensington, MD, UNITED STATES
Arora, Naveen, Delhi, INDIA
Leppla, Stephen H., Bethesda, MD, UNITED STATES
Berzofsky, Jay A., Bethesda, MD, UNITED STATES
PI US 2002048590 A1 20020425

AI US 2001-853530 A1 20010509 (9)
RLI Division of Ser. No. US 1997-937276, filed on 15 Sep 1997, PENDING
PRAI US 1996-25270P 19960917 (60)
DT Utility
FS APPLICATION
LREP TOWNSEND AND TOWNSEND AND CREW, TWO EMBARCADERO CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834
CLMN Number of Claims: 28
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1192
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a vaccine for inducing an immune response in mammal to a specific antigen, where the vaccine comprises a unit dose of a binary toxin **protective antigen** and the **antigen**, which is bound to a binary toxin **protective antigen** binding protein. In one embodiment the vaccine is comprised of an **anthrax protective antigen** and the **antigen** bound to **anthrax protective antigen** binding protein. The present invention also provides a method of immunizing a mammal against an antigen using the vaccine, and a method of inducing antigen-presenting mammalian cells to present specific antigens via the MHC class I processing pathway.

L17 ANSWER 15 OF 33 USPATFULL
AN 2002:72451 USPATFULL
TI Compounds and methods for the treatment and prevention of bacterial infection
IN Collier, R. John, Wellesley, MA, UNITED STATES
Sellman, Bret R., Rochester, NY, UNITED STATES
PI US 2002039588 A1 20020404
AI US 2001-848909 A1 20010504 (9)
PRAI US 2000-201800P 20000504 (60)
DT Utility
FS APPLICATION
LREP CLARK & ELBING LLP, 176 FEDERAL STREET, BOSTON, MA, 02110-2214
CLMN Number of Claims: 28
ECL Exemplary Claim: 1
DRWN 22 Drawing Page(s)
LN.CNT 1502
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides mutant forms of pore-forming toxins. These mutant toxins may be used in vaccines for the prevention of bacterial infection. Additionally, dominant negative mutants may be administered as therapeutics for the treatment of bacterial infection.

L17 ANSWER 16 OF 33 USPATFULL
AN 2002:50802 USPATFULL
TI Computer readable genomic sequence of Haemophilus influenzae Rd, fragments thereof, and uses thereof
IN Fleischmann, Robert D., Gaithersburg, MD, United States
Adams, Mark D., N. Potomac, MD, United States
White, Owen, Gaithersburg, MD, United States
Smith, Hamilton O., Towson, MD, United States
Venter, J. Craig, Potomac, MD, United States
PA Human Genome Sciences, Inc., Rockville, MD, United States (U.S. corporation)
PI US 6355450 B1 20020312
AI US 1995-476102 19950607 (8)
RLI Continuation-in-part of Ser. No. US 1995-426787, filed on 21 Apr 1995, now abandoned
DT Utility

FS GRANTED
EXNAM Primary Examiner: Campell, Bruce R.
CLMN Number of Claims: 88
ECL Exemplary Claim: 1
DRWN 47 Drawing Figure(s); 47 Drawing Page(s)
LN.CNT 4666
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides the sequencing of the entire genome of Haemophilus influenzae Rd, SEQ ID NO: 1. The present invention further provides the sequence information stored on computer readable media, and computer-based systems and methods which facilitate its use. In addition to the entire genomic sequence, the present invention identifies over 1700 protein encoding fragments of the genome and identifies, by position relative to a unique Not I restriction endonuclease site, any regulatory elements which modulate the expression of the protein encoding fragments of the Haemophilus genome.

L17 ANSWER 17 OF 33 USPATFULL
AN 2002:48266 USPATFULL
TI Single target counting assays using semiconductor nanocrystals
IN Empedocles, Stephen Alexander, Mountain View, CA, UNITED STATES
Watson, Andrew R., Belmont, CA, UNITED STATES
Phillips, Vince, Sunnyvale, CA, UNITED STATES
Wong, Edith, Danville, CA, UNITED STATES
PA Quantum Dot Corporation, Hayward, CA, UNITED STATES, 94545 (U.S. corporation)
PI US 2002028457 A1 20020307
AI US 2001-882193 A1 20010613 (9)
RLI Continuation-in-part of Ser. No. US 2001-784866, filed on 15 Feb 2001, PENDING
PRAI US 2000-182844P 20000216 (60)
US 2000-211054P 20000613 (60)
DT Utility
FS APPLICATION
LREP TOWNSEND AND TOWNSEND AND CREW, LLP, TWO EMBARCADERO CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834
CLMN Number of Claims: 18
ECL Exemplary Claim: 1
DRWN 15 Drawing Page(s)
LN.CNT 2844
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides assays that allow for the detection of a single copy of a target of interest. The target species is either directly or indirectly labeled with a semiconductor nanocrytal, "quantum dot." The bright and tunable fluorescence of the quantum dot is readily detected using methods described herein. Also provided are assays that are based on the colocalization of two or more differently colored quantum dots on a single target species, which provides superbly sensitive assays in which the decrease in assay sensitivity caused by non-specific binding of assay mixture components to the assay substrate is minimized. The assays are of use to detect target species including, but are not limited to, nucleic acids, polypeptides, small organic bioactive agents (e.g., drugs, agents of war, herbicides, pesticides, etc.) and organisms.

L17 ANSWER 18 OF 33 USPATFULL
AN 2002:37316 USPATFULL
TI Immuno-adjuvant PDT treatment of metastatic tumors
IN Curry, Patrick Mark, Vancouver, CANADA
Richter, Anna M., Vancouver, CANADA
Levy, Julia G., Vancouver, CANADA
Hunt, David W.C., White Rock, CANADA

PI US 2002022032 A1 20020221
AI US 2001-756687 A1 20010109 (9)
RLI Continuation-in-part of Ser. No. US 2000-556833, filed on 21 Apr 2000, PENDING
PRAI US 1999-130519P 19990423 (60)
DT Utility
FS APPLICATION
LREP MORRISON & FOERSTER LLP, 3811 VALLEY CENTRE DRIVE, SUITE 500, SAN DIEGO, CA, 92130-2332
CLMN Number of Claims: 27
ECL Exemplary Claim: 1
DRWN 4 Drawing Page(s)
LN.CNT 2765
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Immuno-adjuvant photodynamic therapy to treat and prevent metastatic cancer is effected using photosensitizers in combination with immuno-adjuvants to destroy metastatic tumor cells.

L17 ANSWER 19 OF 33 USPATFULL
AN 2002:34423 USPATFULL
TI Noninvasive genetic immunization, expression products therefrom and uses thereof
IN Tang, De-chu C., Birmingham, AL, United States
Marks, Donald H., Rockaway, NJ, United States
Curiel, David T., Birmingham, AL, United States
Shi, Zhongkai, Birmingham, AL, United States
van Kampen, Kent Rigby, Hoover, AL, United States
PA The UAB Research Foundation, Birmingham, AL, United States (U.S. corporation)
PI US 6348450 B1 20020219
AI US 2000-563826 20000503 (9)
RLI Continuation-in-part of Ser. No. US 2000-533149, filed on 23 Mar 2000 Continuation-in-part of Ser. No. US 402527 Continuation-in-part of Ser. No. WO 1998-US16739, filed on 13 Aug 1998
PRAI US 1999-132216P 19990503 (60)
US 1998-75113P 19980211 (60)
US 1997-55520P 19970813 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Crouch, Deborah; Assistant Examiner: Woitach, Joseph T.
LREP Frommer Lawrence & Haug LLP, Frommer, William S., Kowalski, Thomas J.
CLMN Number of Claims: 52
ECL Exemplary Claim: 1
DRWN 21 Drawing Figure(s); 19 Drawing Page(s)
LN.CNT 2393
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed and claimed are methods of non-invasive genetic immunization in an animal and/or methods of inducing a systemic immune or therapeutic response in an animal, products therefrom and uses for the methods and products therefrom. The methods can include contacting skin of the animal with a vector in an amount effective to induce the systemic immune or therapeutic response in the animal. The vector can include and express an exogenous **nucleic acid** molecule encoding an epitope or gene product of interest. The systemic immune response can be to or from the epitope or gene product. The **nucleic acid** molecule can encode an epitope of interest and/or an antigen of interest and/or a **nucleic acid** molecule that stimulates and/or modulates an immunological response and/or stimulates and/or modulates expression, e.g., transcription and/or translation, such as transcription and/or translation of an endogenous and/or exogenous **nucleic acid** molecule; e.g., one or

more of influenza hemagglutinin, influenza nuclear protein, tetanus toxin C-fragment, **anthrax protective antigen** , HIV gp 120, human carcinoembryonic antigen, and/or a therapeutic, an immunomodulatory gene, such as co-stimulatory gene and/or a cytokine gene. The immune response can be induced by the vector expressing the **nucleic acid** molecule in the animal's cells. The immune response can be against a pathogen or a neoplasm. A prophylactic vaccine or a therapeutic vaccine or an immunological composition can include the vector.

L17 ANSWER 20 OF 33 USPATFULL
AN 2001:182107 USPATFULL
TI Vaccine compositions and methods of modulating immune responses
IN Segal, Andrew, Cambridge, MA, United States
PI US 2001031264 A1 20011018
AI US 2001-789922 A1 20010221 (9)
RLI Continuation-in-part of Ser. No. US 1998-7711, filed on 15 Jan 1998, GRANTED, Pat. No. US 6224870
PRAI US 1996-11047P 19960125 (60)
DT Utility
FS APPLICATION
LREP PALMER & DODGE, LLP, ONE BEACON STREET, BOSTON, MA, 02108-3190
CLMN Number of Claims: 7
ECL Exemplary Claim: 1
DRWN 2 Drawing Page(s)
LN.CNT 2512
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides compositions and methods for modulating immune responses in subjects. The invention is based, at least in part, on the discovery that an in-frame translation fusion of an antigen with an APC binding domain of an opsonin forms a molecule, that is, a fusion polypeptide, which when administered to a subject modulates an immune response to the antigen.

L17 ANSWER 21 OF 33 USPATFULL
AN 2001:178820 USPATFULL
TI Organic semiconductor recognition complex and system
IN Kiel, Johnathan L., Universal City, TX, United States
Bruno, John G., San Antonio, TX, United States
Parker, Jill E., Floresville, TX, United States
Alls, John L., San Antonio, TX, United States
Batishko, Charles R., Richland, WA, United States
Holwitt, Eric A., San Antonio, TX, United States
PA Conceptual Mind Works, Inc., San Antonio, TX, United States (U.S. corporation)
PI US 6303316 B1 20011016
AI US 2000-608706 20000630 (9)
PRAI US 1999-142301P 19990702 (60)
US 2000-199620P 20000425 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Horlick, Kenneth R.
LREP Blakely, Sokoloff, Taylor & Zafman
CLMN Number of Claims: 62
ECL Exemplary Claim: 1
DRWN 31 Drawing Figure(s); 15 Drawing Page(s)
LN.CNT 3322
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB In a recognition complex system, **nucleic acid** ligands comprising random **DNA** sequences are operatively coupled to an organic semiconductor and distributed so as to form an array of recognition complexes. When an unknown chemical or biological

analyte is applied to the array, the electrical and/or photochemical properties of one or more of the recognition complexes are altered upon binding of the **nucleic acid** ligand to the analyte. The degree to which the electrical and/or photochemical properties change is a function of the affinity of the **nucleic acid** ligand sequence for the analyte. The electrical and photochemical changes associated with the array, as a whole, can be used as a unique signature to identify the analyte. In certain embodiments, an iterative process of selection and amplification of **nucleic acid** ligands that bind to the analyte can be used to generate a new array with greater affinity and specificity for a target analyte, or to produce one or more **nucleic acid** ligands with high binding affinity for an analyte. The present invention also provides methods for preparing **nucleic acid** ligands that bind with high affinity to an analyte and using such **nucleic acid** ligands to neutralize the analyte.

L17 ANSWER 22 OF 33 USPATFULL
AN 2001:170889 USPATFULL
TI Monocyte-derived dendritic cell subsets
IN Punnonen, Juha, Palo Alto, CA, United States
Chang, Chia-Chun J., Los Gatos, CA, United States
PI US 2001026937 A1 20011004
AI US 2001-760388 A1 20010110 (9)
PRAI US 2000-175552P 20000111 (60)
US 2000-181957P 20000210 (60)
DT Utility
FS APPLICATION
LREP LAW OFFICES OF JONATHAN ALAN QUINE, P O BOX 458, ALAMEDA, CA, 94501
CLMN Number of Claims: 69
ECL Exemplary Claim: 1
DRWN 7 Drawing Page(s)
LN.CNT 3189
AB A novel subset of monocyte-derived dendritic cells are provided. Methods for producing these monocyte-derived dendritic cells and compositions comprising the dendritic cells of the invention are also provided. Methods for inducing an immune response to an antigen of interest using the dendritic cells of the invention are provided. Also provided are methods for therapeutically or prophylactically treating a disease in a subject suffering from the disease using the dendritic cells.

L17 ANSWER 23 OF 33 USPATFULL
AN 2001:67794 USPATFULL
TI Human respiratory syncytial virus peptides with antifusogenic and antiviral activities
IN Barney, Shawn O'Lin, Cary, NC, United States
Lambert, Dennis Michael, Cary, NC, United States
Petteway, Stephen Robert, Cary, NC, United States
PA Trimeris, Inc., Durham, NC, United States (U.S. corporation)
PI US 6228983 B1 20010508
AI US 1995-485264 19950607 (8)
RLI Division of Ser. No. US 1995-470896, filed on 6 Jun 1995
Continuation-in-part of Ser. No. US 1994-360107, filed on 20 Dec 1994
Continuation-in-part of Ser. No. US 1994-255208, filed on 7 Jun 1994
Continuation-in-part of Ser. No. US 1993-73028, filed on 7 Jun 1993, now patented, Pat. No. US 5464933
DT Utility
FS Granted
EXNAM Primary Examiner: Scheiner, Laurie; Assistant Examiner: Parkin, Jeffrey S.
LREP Pennie & Edmonds LLP
CLMN Number of Claims: 62

ECL Exemplary Claim: 1
DRWN 84 Drawing Figure(s); 83 Drawing Page(s)
LN.CNT 32166
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to peptides which exhibit antifusogenic and antiviral activities. The peptides of the invention consist of a 16 to 39 amino acid region of a human respiratory syncytial virus protein. These regions were identified through computer algorithms capable of recognizing the ALLMOTI5, 107x178x4, or PLZIP amino acid motifs. These motifs are associated with the antifusogenic and antiviral activities of the claimed peptides.

L17 ANSWER 24 OF 33 USPATFULL
AN 2001:63248 USPATFULL
TI Vaccine compositions and methods of modulating immune responses
IN Segal, Andrew H., Boston, MA, United States
PA Genitrix, Ltd., Cambridge, MA, United States (U.S. corporation)
PI US 6224870 B1 20010501
AI US 1998-7711 19980115 (9)
RLI Continuation-in-part of Ser. No. US 1997-788143, filed on 24 Jan 1997, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Saunders, David; Assistant Examiner: DeCloux, Amy
LREP Palmer & Dodge, LLP, Williams, Kathleen M.
CLMN Number of Claims: 7
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 2264
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides compositions and methods for modulating immune responses in subjects. The invention is based, at least in part, on the discovery that an in-frame translation fusion of an antigen with an APC binding domain of an opsonin forms a molecule, that is, a fusion polypeptide, which when administered to a subject modulates an immune response to the antigen.

L17 ANSWER 25 OF 33 USPATFULL
AN 2001:56099 USPATFULL
TI Prostate cancer-specific marker
IN French, Cynthia K., Irvine, CA, United States
Schneider, Patrick A., Irvine, CA, United States
Yamamoto, Karen K., San Clemente, CA, United States
PA Diagnostic Products Corporation, Los Angeles, CA, United States (U.S. corporation)
PI US 6218523 B1 20010417
AI US 1998-36315 19980306 (9)
PRAI US 1997-41246P 19970307 (60)
US 1997-47811P 19970515 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Schwartzman, Robert A.; Assistant Examiner: Schmidt, Mary M.
LREP Mueth, Joseph E.
CLMN Number of Claims: 5
ECL Exemplary Claim: 1
DRWN 8 Drawing Figure(s); 6 Drawing Page(s)
LN.CNT 2368
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention provides **cDNA** encoding a prostate-cancer specific marker, Repro-PC-1.0, Repro-PC-1.0 polypeptides and methods for use in diagnosis and therapy.

L17 ANSWER 26 OF 33 USPATFULL
AN 2000:15631 USPATFULL
TI Methods and reagents for inhibiting furin endoprotease
IN Thomas, Gary, Tualatin, OR, United States
Anderson, Eric D., Portland, OR, United States
Thomas, Laurel, Tualatin, OR, United States
Hayflick, Joel S., Seattle, WA, United States
PA Oregan Health Sciences University, Portland, OR, United States (U.S. corporation)
PI US 6022855 20000208
WO 9416073 19940721
AI US 1995-481534 19950914 (8)
WO 1994-US247 19940107
19950914 PCT 371 date
19950914 PCT 102(e) date
RLI Continuation-in-part of Ser. No. US 1993-2202, filed on 8 Jan 1993, now patented, Pat. No. US 5604201
DT Utility
FS Granted
EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Lau, Kawai
LREP McDonnell Boehnen Hulbert & Berghoff
CLMN Number of Claims: 18
ECL Exemplary Claim: 1
DRWN 17 Drawing Figure(s); 10 Drawing Page(s)
LN.CNT 1677
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention relates to method and reagents for inhibiting furin endoprotease activity and specifically for inhibiting furin endoprotease-mediated maturation of bioactive proteins in vivo and in vitro. The invention specifically provides proteins capable of inhibiting furin endoprotease activity. Particularly provided are .alpha..sub.1 -antitrypsin variants that specifically inhibit furin endoprotease activity. Methods for using furin endoprotease inhibition to attenuate or prevent viral protein maturation, and thereby alleviate viral infections, are provided. Also provided are methods for using furin endoprotease inhibition to attenuate or prevent proteolytic processing of bacterial toxins, thereby alleviating bacterial infections. Methods are also provided to inhibit proteolytic processing of biologically active proteins and peptides. The invention also provides pharmaceutically acceptable compositions of therapeutically effective amounts of furin endoprotease inhibitors.

L17 ANSWER 27 OF 33 USPATFULL
AN 2000:9723 USPATFULL
TI Unique nucleotide and amino acid sequence and uses thereof
IN Summers, Max D., Bryan, TX, United States
Braunagel, Sharon C., Bryan, TX, United States
Hong, Tao, Bryan, TX, United States
PA The Texas A & M University System, College Station, TX, United States (U.S. corporation)
PI US 6017734 20000125
AI US 1997-792832 19970130 (8)
RLI Continuation-in-part of Ser. No. US 1996-678435, filed on 3 Jul 1996, now abandoned
PRAI US 1995-955P 19950707 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Elliott, George C.; Assistant Examiner: Schwartzman, Robert
LREP Arnold, White & Durkee
CLMN Number of Claims: 56

ECL Exemplary Claim: 1
DRWN 47 Drawing Figure(s); 24 Drawing Page(s)
LN.CNT 7846
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Provided are hydrophobic targeting sequences, which may serve to target heterologous proteins to a variety of cellular membranes. In particular, the structural components of the nuclear envelope, or those components which become nucleus-associated, may be targeted with the sequences provided. Also provided are methods of targeting heterologous proteins to particular membranes, and the use of these targeted proteins in therapeutic, diagnostic and insecticidal applications.

L17 ANSWER 28 OF 33 USPATFULL
AN 1999:141912 USPATFULL
TI Compositions and methods for delivery of genetic material
IN Weiner, David B., Merion, PA, United States
Williams, William V., Havertown, PA, United States
Wang, Bin, Havertown, PA, United States
PA The Trustees of The University of Pennsylvania, Philadelphia, PA, United States (U.S. corporation)
The Wistar Institute, Philadelphia, PA, United States (U.S. corporation)
PI US 5981505 19991109
WO 9416737 19940804
AI US 1997-979385 19971126 (8)
WO 1994-US899 19940126
19950828 PCT 371 date
19950828 PCT 102(e) date
RLI Continuation-in-part of Ser. No. US 1993-124962, filed on 21 Sep 1993, now abandoned And a continuation-in-part of Ser. No. US 1993-93235, filed on 15 Jul 1993, now abandoned And a continuation of Ser. No. US 1995-495684, filed on 28 Aug 1995, now abandoned which is a continuation-in-part of Ser. No. US 1993-125012, filed on 21 Sep 1993, now patented, Pat. No. US 5593972, issued on 14 Jan 1997 which is a continuation-in-part of Ser. No. US 1993-29336, filed on 11 Mar 1993, now abandoned which is a continuation-in-part of Ser. No. US 1993-8342, filed on 26 Jan 1993, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Railey, II, Johnny F.
LREP Woodcock Washburn Kurtz Mackiewicz & Norris LLP
CLMN Number of Claims: 75
ECL Exemplary Claim: 1
DRWN 23 Drawing Figure(s); 12 Drawing Page(s)
LN.CNT 4084
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods of inducing genetic material into cells of an individual and compositions and kits for practicing the same are disclosed. The methods comprise the steps of contacting cells of an individual with a **polynucleotide** function enhancer and administering to the cells, a **nucleic acid** molecule that is free of retroviral particles. The **nucleic acid** molecule comprises a nucleotide sequence that encodes a protein that comprises at least one epitope that is identical or substantially similar to an epitope of a pathogen antigen or an antigen associated with a hyperproliferative or autoimmune disease, a protein otherwise missing from the individual due to a missing, non-functional or partially functioning gene, or a protein that produces a therapeutic effect on an individual. Methods of prophylactically and therapeutically immunizing an individual against HIV are disclosed. Pharmaceutical compositions and kits for practicing methods of the present invention are disclosed.

L17 ANSWER 29 OF 33 USPATFULL

AN 1999:141305 USPATFULL
TI Adjuvant for transcutaneous immunization
IN Glenn, Gregory M., Bethesda, MD, United States
Alving, Carl R., Bethesda, MD, United States
PA The United States of America as represented by the U.S. Army Medical Research & Material Command, Washington, DC, United States (U.S. government)
PI US 5980898 19991109
AI US 1997-896085 19970717 (8)
RLI Continuation-in-part of Ser. No. US 1996-749164, filed on 14 Nov 1996
DT Utility
FS Granted
EXNAM Primary Examiner: Saunders, David; Assistant Examiner: Tung, Mary Beth
LREP Pillsbury, Madison & Sutro LLP
CLMN Number of Claims: 13
ECL Exemplary Claim: 1,11
DRWN 1 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 1988
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A transcutaneous immunization system delivers antigen to immune cells without perforation of the skin, and induces an immune response in an animal or human. The system uses an adjuvant, preferably an ADP-ribosylating exotoxin, to induce an antigen-specific immune response (e.g., humoral and/or cellular effectors) after transcutaneous application of a formulation containing antigen and adjuvant to intact skin of the animal or human. The efficiency of immunization may be enhanced by adding hydrating agents (e.g., liposomes), penetration enhancers, or occlusive dressings to the transcutaneous delivery system. This system may allow activation of Langerhans cells in the skin, migration of the Langerhans cells to lymph nodes, and antigen presentation.

L17 ANSWER 30 OF 33 USPATFULL
AN 1999:109966 USPATFULL
TI Opsonin-enhanced cells, and methods of modulating an immune response to an antigen
IN Segal, Andrew H., Boston, MA, United States
PA Whitenead Institute for Biomedical Research, Cambridge, MA, United States (U.S. corporation)
PI US 5951976 19990914
AI US 1997-826259 19970327 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Hutzell, Paula K.; Assistant Examiner: Bansal, Geetha P.
LREP Banner & Witcoff, Ltd.
CLMN Number of Claims: 22
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 2180
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are methods and compositions wherein opsonin-enhanced cells, that is, cells which have been 1) modified so as to express an opsonin from a recombinant **nucleic acid**, 2) modified so as to express higher levels of an endogenous opsonin, or 3) mixed with an exogenous opsonin, when administered to a subject, modulate the immune response in the recipient to a selected antigen or antigens contained in or attached to the cells.

L17 ANSWER 31 OF 33 USPATFULL
AN 97:94207 USPATFULL
TI **Anthrax** toxin fusion proteins and related methods

IN Leppla, Stephen H., Bethesda, MD, United States
Klimpel, Kurt R., Gaithersburg, MD, United States
Arora, Naveen, Delhi, India
Singh, Yogendra, Delhi, India
Nichols, Peter J., Welling Kent, United Kingdom
PA The Government of the United States as represented by the Secretary of the Department of Health and Human Services, Washington, DC, United States (U.S. government)
PI US 5677274 19971014
AI US 1993-82849 19930625 (8)
RLI Continuation-in-part of Ser. No. US 1993-21601, filed on 12 Feb 1993, now patented, Pat. No. US 5591631
DT Utility
FS Granted
EXNAM Primary Examiner: Jagannathan, Vasu S.; Assistant Examiner: Romeo, David S.
LREP Townsend and Townsend and Crew
CLMN Number of Claims: 12
ECL Exemplary Claim: 1
DRWN 1 Drawing Figure(s); 1 Drawing Page(s)
LN.CNT 3382
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a **nucleic acid** encoding a fusion protein comprising a nucleotide sequence encoding the **anthrax protective antigen** (PA) binding domain of the native **anthrax** lethal factor (LF) protein and a nucleotide sequence encoding an activity inducing domain of a second protein. Also provided is a **nucleic acid** encoding a fusion protein comprising a nucleotide sequence encoding the translocation domain and LF binding domain of the native **anthrax** PA protein and a nucleotide sequence encoding a ligand domain which specifically binds a cellular target. Proteins encoded by the **nucleic acid** of the invention, vectors comprising the nucleic acids and hosts capable of expressing the protein encoded by the nucleic acids are also provided. A composition comprising the PA binding domain of the native LF protein chemically attached to a non-LF activity inducing moiety is further provided. A method for delivering an activity to a cell is provided. The steps of the method include a) administering to the cell a protein comprising the translocation domain and the LF binding domain of the native PA protein and a ligand domain, and b) administering to the cell a product comprising the PA binding domain of the native LF protein and a non-LF activity inducing moiety, whereby the product administered in step b) is internalized into the cell and performs the activity within the cell. The invention also provides proteins including an **anthrax protective antigen** which has been mutated to replace the trypsin cleavage site with residues recognized specifically by the HIV-1 protease.

L17 ANSWER 32 OF 33 USPATFULL
AN 97:14677 USPATFULL
TI Methods and reagents for inhibiting furin endoprotease
IN Thomas, Gary, Tualatin, OR, United States
Anderson, Eric D., Portland, OR, United States
Thomas, Laurel, Tualatin, OR, United States
Hayflick, Joel S., Seattle, WA, United States
PA State of Oregon, Acting by and through the Oregon State Board of Higher Education on Behalf of the Oregon Health Sciences University, a non-profit organization, Portland, OR, United States (U.S. corporation)
PI US 5604201 19970218
AI US 1993-2202 19930108 (8)
DT Utility
FS Granted

EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Lau, Kawai
LREP Banner & Allegretti, Ltd.
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 6 Drawing Figure(s); 6 Drawing Page(s)
LN.CNT 1307
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention relates to methods and reagents for inhibiting furin endoprotease activity and specifically for inhibiting furin endoprotease-mediated maturation of bioactive proteins in vivo and in vitro. The invention specifically provides proteins capable of inhibiting furin endoprotease activity. Particularly provided are .alpha..sub.1 -antitrypsin variants that specifically inhibit furin endoprotease activity. Methods for using furin endoprotease inhibition to attenuate or prevent viral protein maturation, and thereby alleviate viral infections, are provided. Also provided are methods for using furin endoprotease inhibition to attenuate or prevent proteolytic processing of bacterial toxins, thereby alleviating bacterial infections. Methods are also provided to inhibit proteolytic processing of biologically active proteins and peptides. The invention also provides pharmaceutically acceptable compositions of therapeutically effective amounts of furin endoprotease inhibitors.

L17 ANSWER 33 OF 33 USPATFULL
AN 97:1356 USPATFULL
TI **Anthrax** toxin fusion proteins, **nucleic acid** encoding same
IN Leppla, Stephen H., Bethesda, MD, United States
Klimpel, Kurt R., Gaithersburg, MD, United States
Arora, Naveen, Delhi, India
Singh, Yogendra, Delhi, India
Nicholls, Peter J., Welling Kent, United Kingdom
PA The United States of America as represented by the Department of Health and Human Services, Washington, DC, United States (U.S. government)
PI US 5591631 19970107
AI US 1993-21601 19930212 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Walsh, Stephen G.
LREP Townsend and Townsend and Crew
CLMN Number of Claims: 13
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 2181
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a **nucleic acid** encoding a fusion protein, comprising a nucleotide sequence encoding the **protective antigen** (PA) binding domain of the native lethal factor (LF) protein and a nucleotide sequence encoding an activity inducing domain of a second protein. Also provided is a **nucleic acid** encoding a fusion protein, comprising a nucleotide sequence encoding the translocation domain and LF binding domain of the native PA protein and a nucleotide sequence encoding a ligand domain which specifically binds a cellular target. Proteins encoded by the **nucleic acid** of the invention, vectors comprising the nucleic acids and hosts capable of expressing the protein encoded by the nucleic acids are also provided. A composition comprising the PA binding domain of the native LF protein chemically attached to a non-LF activity inducing moiety is further provided. A method for delivering an activity to a cell is provided. The steps of the method include administering to the cell a protein comprising the translocation domain and the LF binding domain of the native PA protein

and a ligand domain, and administering to the cell a product comprising the PA binding domain of the native LF protein and a non-LF activity inducing moiety, whereby the product administered is internalized into the cell and performs the activity within the cell.

In solution or when bound to receptors on Chinese hamster ovary K1 cells, neither mutant alone bound ligand, but a mixture of them did. After the two mutants were proteolytically activated and mixed with ligand in solution, a ternary complex was isolated containing one molecule of each protein. Thus EF and LF bind stably only to PA63 dimers or higher order oligomers. These findings are relevant to the kinetics and pathways of assembly of **anthrax** toxin complexes.

L11 ANSWER 5 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 4
AN 2002:168880 BIOSIS
DN PREV200200168880
TI Mapping the **anthrax protective antigen** binding site on the lethal and edema factors.
AU Lacy, D. Borden; Mourez, Michael; Fouassier, Alexandre; **Collier, R. John (1)**
CS (1) Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA, 02115: jcollier@hms.harvard.edu USA
SO Journal of Biological Chemistry, (January 25, 2002) Vol. 277, No. 4, pp. 3006-3010. http://www.jbc.org/. print.
ISSN: 0021-9258.
DT Article
LA English
AB Entry of **anthrax** edema factor (EF) and lethal factor (LF) into the cytosol of eukaryotic cells depends on their ability to translocate across the endosomal membrane in the presence of **anthrax protective antigen** (PA). Here we report attributes of the N-terminal domains of EF and LF (EFN and LFN, respectively) that are critical for their initial interaction with PA. We found that deletion of the first 36 residues of LFN had no effect on its binding to PA or its ability to be translocated. To map the binding site for PA, we used the three-dimensional structure of LF and sequence similarity between EF and LF to select positions for mutagenesis. We identified seven sites in LFN (Asp-182, Asp-187, Leu-188, Tyr-223, His-229, Leu-235, and Tyr-236) where mutation to Ala produced significant binding defects, with H229A and Y236A almost completely eliminating binding. Homologous mutants of EFN displayed nearly identical defects. Cytotoxicity assays confirmed that the LFN mutations impact intoxication. The seven mutation-sensitive amino acids are clustered on the surface of LF and form a small convoluted patch with both hydrophobic and hydrophilic character. We propose that this patch constitutes the recognition site for PA.

L11 ANSWER 6 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 5
AN 2002:184710 BIOSIS
DN PREV200200184710
TI PA63 channel of **anthrax** toxin: An extended beta-barrel.
AU Nassi, Shilla (1); **Collier, R. John;** Finkelstein, Alan (1)
CS (1) Department of Neuroscience and Department of Physiology and Biophysics, Albert Einstein College of Medicine, 1300 Morris Park Avenue, Bronx, NY, 10461 USA
SO Biochemistry, (February 5, 2002) Vol. 41, No. 5, pp. 1445-1450. http://pubs.acs.org/journals/bichaw/. print.
ISSN: 0006-2960.
DT Article
LA English
AB **Anthrax** toxin consists of three protein components: **protective antigen** (PA), lethal factor (LF), and edema factor (EF). PA63, generated by protease "nicking" of whole PA, is responsible for delivering the toxin's catalytic fragments (LF and EF) to the target cell's cytosol. In planar bilayer membranes, trypsin-nicked PA makes cation-selective voltage-gated channels with a pore diameter of

critical in the pathogenesis of **anthrax**. It is a highly specific protease that cleaves members of the mitogen-activated protein kinase kinase (MAPKK) family near to their amino termini, leading to the inhibition of one or more signalling pathways. Here we describe the crystal structure of LF and its complex with the N terminus of MAPKK-2. LF comprises four domains: domain I binds the membrane-translocating component of **anthrax** toxin, the **protective antigen** (PA); domains II, III and IV together create a long deep groove that holds the 16-residue N-terminal tail of MAPKK-2 before cleavage. Domain II resembles the ADP-ribosylating toxin from Bacillus cereus, but the active site has been mutated and recruited to augment substrate recognition. Domain III is inserted into domain II, and seems to have arisen from a repeated duplication of a structural element of domain II. Domain IV is distantly related to the zinc metalloprotease family, and contains the catalytic centre; it also resembles domain I. The structure thus reveals a protein that has evolved through a process of gene duplication, mutation and fusion, into an enzyme with high and unusual specificity.

L11 ANSWER 17 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 11
AN 2001:566770 BIOSIS
DN PREV200100566770
TI Identification of the cellular receptor for **anthrax** toxin.
AU **Bradley, Kenneth A.; Mogridge, Jeremy;** Mourez, Michael; **Collier, R. John; Young, John A. T. (1)**
CS (1) McArdle Laboratory for Cancer Research, University of Wisconsin-Madison, 1400 University Avenue, Madison, WI, 53706: young@oncology.wisc.edu USA
SO Nature (London), (8 November, 2001) Vol. 414, No. 6860, pp. 225-229. print.
ISSN: 0028-0836.
DT Article
LA English
SL English
AB The tripartite toxin secreted by Bacillus **anthracis**, the causative agent of **anthrax**, helps the bacterium evade the immune system and can kill the host during a systemic infection. Two components of the toxin enzymatically modify substrates within the cytosol of mammalian cells: oedema factor (OF) is an adenylate cyclase that impairs host defences through a variety of mechanisms including inhibiting phagocytosis; lethal factor (LF) is a zinc-dependent protease that cleaves mitogen-activated protein kinase kinase and causes lysis of macrophages. **Protective antigen** (PA), the third component, binds to a cellular receptor and mediates delivery of the enzymatic components to the cytosol. Here we describe the cloning of the human PA receptor using a genetic complementation approach. The receptor, termed ATR (**anthrax** toxin receptor), is a type I membrane protein with an extracellular von Willebrand factor A domain that binds directly to PA. In addition, a soluble version of this domain can protect cells from the action of the toxin.

L11 ANSWER 18 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 12
AN 2000:393065 BIOSIS
DN PREV200000393065
TI A quantitative study of the interactions of Bacillus **anthracis** edema factor and lethal factor with activated **protective antigen**.
AU Elliott, Jennifer L.; **Mogridge, Jeremy; Collier, R. John (1)**
CS (1) Department of Microbiology and Molecular Genetics, Harvard Medical

School, Boston, MA, 02115 USA
SO Biochemistry, (June 6, 2000) Vol. 39, No. 22, pp. 6706-6713. print.
ISSN: 0006-2960.
DT Article
LA English
SL English
AB Bacillus **anthracis** secretes three proteins, which associate in binary combinations to form toxic complexes at the surface of mammalian cells. Receptor-bound **protective antigen** (PA) is proteolytically activated, yielding a 63 kDa fragment (PA63). PA63 oligomerizes into heptamers, which bind edema factor (EF) or lethal factor (LF) to form the toxic complexes. We undertook a quantitative analysis of the interactions of EF with PA63 by means of surface plasmon resonance (SPR) measurements. Heptameric PA63 was covalently bound by amine coupling to an SPR chip, or noncovalently bound via a C-terminal hexahistidine tag on the protein to Ni2+nitrilotriacetate groups on the chip. Values of kon and koff for EF at 23 degreeC were apprx3 X 105 M-1 s-1 and (3-5) X 10-4 s-1, respectively, giving a calculated Kd of (1-2) X 10-9 M. A similar value of Kd (7 X 10-10 M) was obtained when we measured the binding of radiolabeled EF to receptor-bound PA63 on the surface of L6 cells (at 4 degreeC). Each of these analyses was also performed with LF and LFN (the terminal 255 residues of LF), and values obtained were comparable to those for EF. The similarity in the dissociation constants determined by SPR and by measurements on the cell surface suggests that the presence of the receptor does not play a large role in the interaction between PA63 and EF/LF.

L11 ANSWER 19 OF 44 CAPLUS COPYRIGHT 2002 ACS
AN 2000:568809 CAPLUS
DN 133:262508
TI Proteolytic activation of receptor-bound **anthrax protective antigen** on macrophages promotes its internalization
AU Beauregard, Kathryn E.; **Collier, R. John**; Swanson, Joel A.
CS Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA, 02115, USA
SO Cellular Microbiology (2000), 2(3), 251-258
CODEN: CEMIF5; ISSN: 1462-5814
PB Blackwell Science Ltd.
DT Journal
LA English
AB Immunofluorescence and other methods have been used to probe the self-assembly and internalization of the binary toxin, **anthrax** lethal toxin (LeTx), in primary murine macrophages. Proteolytic activation of **protective antigen** (PA; 83 kDa, the B moiety of the toxin) by furin was the rate-limiting step in internalization of LeTx and promoted clearance of PA from the cell surface. A furin-resistant form of PA remained at the cell surface for at least 90 min. Oligomerization of receptor-bound PA63, the 63 kDa active fragment of PA, was manifested by its conversion to a pronase-resistant state, characteristic of the heptameric prepore form in soln. That oligomerization of PA63 triggers toxin internalization is supported by the observation that PA20, the complementary 20 kDa fragment of PA, inhibited clearance of nicked PA. The PA63 prepore, with or without lethal factor (LF), cleared slowly from the cell surface. These studies show that proteolytic cleavage of PA, in addn. to permitting oligomerization and LF binding, also promotes internalization of the protein. The relatively long period of activation and internalization of PA at the cell surface may reflect adaptation of this binary toxin that maximizes self-assembly.
RE.CNT 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

response, indicating that this molecule functioned similarly to the genetically fused forms used previously. We also report the results of an analysis of two aspects of this system important for the development of experimental vaccines. First, CD4 knockout mice were unable to generate a CTL response when treated with PA plus an LFn-epitope fusion protein, suggesting that CD4+ helper responses are essential for stimulating specific CTL with the PA-LFn system. Second, we now show that primary injection with this system does not generate any detectable antibody response to the vaccine components and that prior immunization has no effect on priming a CTL response to an unrelated epitope upon subsequent injection.

L11 ANSWER 28 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 18
AN 1998:205886 BIOSIS
DN PREV199800205886
TI Identification of residues lining the **anthrax protective antigen** channel.
AU Benson, Ericka L.; Huynh, Paul D.; Finkelstein, Alan; **Collier, R. John (1)**
CS (1) Dep. Microbiol. Mol. Genet., Harvard Med. Sch., 200 Longwood Ave., Boston, MA 02115 USA
SO Biochemistry, (March 17, 1998) Vol. 37, No. 11, pp. 3941-3948. ISSN: 0006-2960.
DT Article
LA English
AB In its activated 63 kDa form, the **protective antigen** (PA) component of **anthrax** toxin forms a heptameric prepore, which converts to a pore (channel) in endosomal membranes at low pH and mediates translocation of the toxin's enzymic moieties to the cytosol. It has been proposed that the prepore-to-pore conversion involves a conformational rearrangement of a disordered amphipathic loop (D2L2; residues 302-325), in which loops from the 7 protomers combine to form a transmembrane 14-stranded beta barrel. To test this model, we generated Cys substitutions in 24 consecutive residues of the D2L2 loop, formed channels in artificial bilayers with each mutant, and examined changes in channel conductance after adding the thiol-reactive, bilayer-impermeant reagent methanethiosulfonate ethyltrimethylammonium (MTS-ET) to the trans compartment. The rationale for these experiments is that reaction of MTS-ET with a Cys residue adds a positively charged group and therefore would likely reduce channel conductance if the residue were in the ion-conducting pathway. We found alternating reduction and absence of reduction of conductance in consecutive residues over two stretches (residues 302-311 and 316-325). This pattern is consistent with alternating polar and apolar residues of the two stretches projecting into the pore lumen and into the bilayer, respectively. Residues connecting these two stretches (residues 312-315) were responsive to MTS-ET, consistent with their being in a turn region. Single channels formed by selected mutants (H304C and N306C) showed multiple conductance step changes in response to MTS-ET, consistent with an oligomeric pore. We also found that the binding site for the channel-blocking tetraalkylammonium ions is located cis relative to the inserted D2L2 loops. These findings constitute strong evidence in favor of the model of conversion of the prepore to a 14-stranded beta barrel pore and solidify the foundation for studies to understand the mechanism of translocation by **anthrax** toxin.

L11 ANSWER 29 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 19
AN 1998:125927 BIOSIS
DN PREV199800125927
TI **Anthrax** toxin-mediated delivery in vivo and in vitro of a

8371-8376. print.
ISSN: 0021-9258.
DT Article
LA English
SL English

L12 ANSWER 5 OF 13 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2001:157442 BIOSIS
DN PREV200100157442
TI Involvement of domain 3 in oligomerization by the **protective antigen** moiety of **anthrax** toxin.
AU **Mogridge, Jeremy**; Mourez, Michael; **Collier, R. John (1)**
CS (1) Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA, 02115: jcollier@hms.harvard.edu USA
SO Journal of Bacteriology, (March, 2001) Vol. 183, No. 6, pp. 2111-2116. print.
ISSN: 0021-9193.
DT Article
LA English
SL English

L12 ANSWER 6 OF 13 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2000:393065 BIOSIS
DN PREV200000393065
TI A quantitative study of the interactions of Bacillus **anthracis** edema factor and lethal factor with activated **protective antigen**.
AU Elliott, Jennifer L.; **Mogridge, Jeremy**; **Collier, R. John (1)**
CS (1) Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA, 02115 USA
SO Biochemistry, (June 6, 2000) Vol. 39, No. 22, pp. 6706-6713. print.
ISSN: 0006-2960.
DT Article
LA English
SL English

L12 ANSWER 7 OF 13 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1999:338648 BIOSIS
DN PREV199900338648
TI **Anthrax** toxin entry into polarized epithelial cells.
AU Beauregard, Kathryn E.; Wimer-Mackin, Susan; **Collier, R. John**; Lencer, Wayne I. (1)
CS (1) GI Cell Biology, Children's Hospital, 300 Longwood Ave., Enders 1220, Boston, MA, 02115 USA
SO Infection and Immunity, (June, 1999) Vol. 67, No. 6, pp. 3026-3030.
ISSN: 0019-9567.
DT Article
LA English
SL English

L12 ANSWER 8 OF 13 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1997:158460 BIOSIS
DN PREV199799457663
TI Crystal structure of the **anthrax** toxin **protective antigen**.
AU Petosa, Carlo (1); **Collier, R. John**; Klimpel, Kurt R.; Leppla, Stephen H.; Liddington, Robert C.
CS (1) Biochemistry Dep., Univ. Leicester, Leicester LE1 7RH UK
SO Nature (London), (1997) Vol. 385, No. 6619, pp. 833-838.
ISSN: 0028-0836.
DT Article

L16 ANSWER 16 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 2
AN 2002:452545 BIOSIS
DN PREV200200452545
TI Identification of amino acid residues of **anthrax protective antigen** involved in binding with lethal factor.
AU Chauhan, Vibha; Bhatnagar, Rakesh (1)
CS (1) Centre for Biotechnology, Jawaharlal Nehru University, New Delhi, 110067: rakeshb01@hotmail.com India
SO Infection and Immunity, (August, 2002) Vol. 70, No. 8, pp. 4477-4484. print.
ISSN: 0019-9567.
DT Article
LA English

L16 ANSWER 17 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2002:364347 BIOSIS
DN PREV200200364347
TI 2001: A year of major advances in **anthrax** toxin research.
AU Mourez, Michael (1); Lacy, D. Borden (1); Cunningham, Kristina (1); Legmann, Rachel (1); Sellman, Bret R.; Mogridge, Jeremy; Collier, R. John (1)
CS (1) Dept of Microbiology and Molecular Genetics, Harvard Medical School, 200 Longwood Ave, Boston, MA, 02115: jcollier@hms.harvard.edu USA
SO Trends in Microbiology, (June, 2002) Vol. 10, No. 6, pp. 287-293. http://journals.bmn.com/journals/list/latest?jcode=tim. print.
ISSN: 0966-842X.
DT General Review
LA English

L16 ANSWER 18 OF 123 CAPLUS COPYRIGHT 2002 ACS
AN 2002:562946 CAPLUS
TI Progress on the **anthrax** toxin and the cellular clone **receptor** for **anthrax** toxin
AU Liu, Cheng-yi; Li, Yan-ling; Duan, Rui; Li, Yan; Cai, Xiong-wei; Huang, Ping
CS The Information Biology Group of Laboratory of Light Transmission Optics, South China Normal University, Canton, 510631, Peop. Rep. China
SO Huanan Shifan Daxue Xuebao, Ziran Kexueban (2002), (2), 114-119
CODEN: HSDZER; ISSN: 1000-5463
PB Huanan Shifan Daxue
DT Journal
LA Chinese

L16 ANSWER 19 OF 123 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 2002280320 EMBASE
TI Structure and function of **anthrax** toxin.
AU Lacy D.B.; Collier R.J.
CS D.B. Lacy, Department of Microbiology, Harvard Medical School, Boston, MA 02115, United States. jcollier@hms.harvard.edu
SO Current Topics in Microbiology and Immunology, (2002) 271/- (61-85).
Refs: 83
ISSN: 0070-217X CODEN: CTMIA3
CY Germany
DT Journal; General Review
FS 004 Microbiology
026 Immunology, Serology and Transplantation
LA English
SL English

L16 ANSWER 20 OF 123 BIOTECHDS COPYRIGHT 2002 THOMSON DERWENT AND ISI

DUPLICATE 5
AN 2001:238485 BIOSIS
DN PREV200100238485
TI Point mutations in **anthrax protective antigen** that block translocation.
AU Sellman, Bret R.; Nassi, Shilla; Collier, R. John (1)
CS (1) Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA, 02115: jcollier@hms.harvard.edu USA
SO Journal of Biological Chemistry, (March 16, 2001) Vol. 276, No. 11, pp. 8371-8376. print.
ISSN: 0021-9258.
DT Article
LA English
SL English

L16 ANSWER 33 OF 123 MEDLINE DUPLICATE 6
AN 2001503900 MEDLINE
DN 21437664 PubMed ID: 11553601
TI Purification of **anthrax** edema factor from Escherichia coli and identification of residues required for binding to **anthrax protective antigen**.
AU Kumar P; Ahuja N; Bhatnagar R
CS Centre for Biotechnology, Jawaharlal Nehru University, New Delhi 110067, India.
SO INFECTION AND IMMUNITY, (2001 Oct) 69 (10) 6532-6.
Journal code: 0246127. ISSN: 0019-9567.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200110
ED Entered STN: 20010913
Last Updated on STN: 20011029
Entered Medline: 20011025

L16 ANSWER 34 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 7
AN 2001:157442 BIOSIS
DN PREV200100157442
TI Involvement of domain 3 in oligomerization by the **protective antigen** moiety of **anthrax** toxin.
AU Mogridge, Jeremy; Mourez, Michael; Collier, R. John (1)
CS (1) Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA, 02115: jcollier@hms.harvard.edu USA
SO Journal of Bacteriology, (March, 2001) Vol. 183, No. 6, pp. 2111-2116. print.
ISSN: 0021-9193.
DT Article
LA English
SL English

L16 ANSWER 35 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 8
AN 2001:570431 BIOSIS
DN PREV200100570431
TI Know thine enemy.
AU Smith, Orla
SO Science (Washington D C), (9 November, 2001) Vol. 294, No. 5545, pp. 1298. print.
ISSN: 0036-8075.
DT General Review
LA English

DT Conference
LA English

L11 ANSWER 9 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2002:364347 BIOSIS
DN PREV200200364347
TI 2001: A year of major advances in **anthrax** toxin research.
AU Mourez, Michael (1); Lacy, D. Borden (1); Cunningham, Kristina (1); Legmann, Rachel (1); Sellman, Bret R.; **Mogridge, Jeremy; Collier, R. John (1)**
CS (1) Dept of Microbiology and Molecular Genetics, Harvard Medical School, 200 Longwood Ave, Boston, MA, 02115: jcollier@hms.harvard.edu USA
SO Trends in Microbiology, (June, 2002) Vol. 10, No. 6, pp. 287-293. http://journals.bmn.com/journals/list/latest?jcode=tim. print. ISSN: 0966-842X.
DT General Review
LA English
AB **Anthrax** is caused when spores of Bacillus **anthracis** enter a host and germinate. The bacteria multiply and secrete a tripartite toxin causing local edema and, in systemic infection, death. In nature, **anthrax** is primarily observed in cattle and other herbivores; humans are susceptible but rarely affected. In 2001, **anthrax** spores were used effectively for the first time in bioterrorist attacks, resulting in 11 confirmed cases of human disease and five deaths. These events have underscored the need for improved prophylaxis, therapeutics and a molecular understanding of the toxin. The good news about **anthrax** is that several decisive discoveries regarding the toxin have been reported recently. Most notably, the toxin receptor was identified, the 3-D structures of two of the toxin subunits were solved and potent in vivo inhibitors were designed. These findings have improved our understanding of the intoxication mechanism and are stimulating the design of strategies to fight disease in the future.

L11 ANSWER 10 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2002:301231 BIOSIS
DN PREV200200301231
TI The PA63 channel of **anthrax** toxin: An extended beta-barrel.
AU Nassi, Shilla (1); Finkelstein, Alan (1); **Collier, R. John**
CS (1) Albert Einstein College of Medicine, 1300 Morris Park Ave, Bronx, NY, 10461 USA
SO Biophysical Journal, (January, 2002) Vol. 82, No. 1 Part 2, pp. 195a. http://intl.biophysj.org/. print.
Meeting Info.: 46th Annual Meeting of the Biophysical Society San Francisco, California, USA February 23-27, 2002
ISSN: 0006-3495.
DT Conference
LA English

L11 ANSWER 11 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 7
AN 2001:238485 BIOSIS
DN PREV200100238485
TI Point mutations in **anthrax protective antigen** that block translocation.
AU Sellman, Bret R.; Nassi, Shilla; **Collier, R. John (1)**
CS (1) Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA, 02115: jcollier@hms.harvard.edu USA
SO Journal of Biological Chemistry, (March 16, 2001) Vol. 276, No. 11, pp. 8371-8376. print.
ISSN: 0021-9258.
DT Article
LA English

ISSN: 0065-7727.
DT Conference
LA English

L11 ANSWER 40 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 25
AN 1994:437552 BIOSIS
DN PREV199497450552
TI **Anthrax Protective Antigen** Forms Oligomers during Intoxication of mammalian Cells.
AU Milne, Jill C.; Furlong, Deirdre; Hanna, Philip C.; Wall, Joseph S.; **Collier, R. John (1)**
CS (1) Dep. Microbiol. Mol. Genet., Shipley Inst. Med., Harvard Med. Sch., Boston, MA 02115 USA
SO Journal of Biological Chemistry, (1994) Vol. 269, No. 32, pp. 20607-20612. ISSN: 0021-9258.
DT Article
LA English
AB The **protective antigen** component (PA) of **anthrax** toxin binds to receptors on target cells and conveys the toxin's edema factor (EF) and lethal factor (LF) components into the cytoplasm. PA (83 kDa) is processed by a cellular protease, yielding a 63-kDa fragment (PA-63), which binds EF and/or LF. When exposed to acidic pH, PA-63 inserts into membranes and forms ion-conductive channels. By electron microscopy, a significant fraction of purified PA-63 was found to be in the form of a multisubunit ring-shaped oligomer (outer diameter, 10.4 nm). The rings are heptameric, as judged by inspection and by rotational power spectra. Purified PA-63 showed a high Mr band, apparently corresponding to the oligomer, on SDS-polyacrylamide gels, and oligomer of similar size was formed in cells in a time-dependent manner after addition of full-length PA. Inhibitors of internalization and endosome acidification blocked conversion of cell-associated PA to a high molecular weight species, and medium at pH 5.0 induced oligomer formation in the presence or absence of the inhibitors. These results correlate PA-63 oligomerization with conditions required for translocation of EF and LF across lipid bilayers, implying that the PA-63 oligomer may function in translocation.

L11 ANSWER 41 OF 44 LIFESCI COPYRIGHT 2002 CSA
AN 95:110089 LIFESCI
TI Effect of **anthrax** toxin's lethal factor on ion channels formed by the **protective antigen**
AU Zhao, Jianmin; Milne, J.C.; **Collier, R.J.***
CS Dep. Microbiol. and Mol. Genet. and Shipley Inst. Med., Harvard Med. Sch., Boston, MA 02115, USA
SO J. BIOL. CHEM., (1994) vol. 270, no. 31, pp. 18626-18630. ISSN: 0021-9258.
DT Journal
FS X
LA English
SL English
AB **Protective antigen** (PA), a component of **anthrax** toxin, mediates translocation of the toxin's lethal and edema factors (LF and EF, respectively) to the cytoplasm, via a pathway involving their release from an acidic intracellular compartment. PA sub(63), a 63-kDa proteolytic fragment of PA, can be induced to form ion-conductive channels in the plasma membrane of mammalian cells by acidification of the medium. These channels are believed to be comprised of dodecyl sulfate-resistant oligomers (heptameric rings) of PA sub(63) seen by electron microscopy of the purified protein. Here we report that the PA sub(63)-mediated efflux of super(86)Rb super(+) from preloaded CHO-K1 cells under acidic conditions is strongly inhibited (greater than

LA English

L12 ANSWER 9 OF 13 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1995:203456 BIOSIS
DN PREV199598217756
TI **Protective antigen**-binding domain of **anthrax** lethal factor mediates translocation of a heterologous protein fused to its amino- or carboxy-terminus.
AU Milne, Jill C.; Blanke, Steven R.; Hanna, Philip C.; **Collier, R. John (1)**
CS (1) Dep. Microbiol. Mol. Genetics, Shipley Inst. Med., Harvard Med. Sch., Boston, MA 02115 USA
SO Molecular Microbiology, (1995) Vol. 15, No. 4, pp. 661-666. ISSN: 0950-382X.
DT Article
LA English

L12 ANSWER 10 OF 13 CAPLUS COPYRIGHT 2002 ACS
AN 2002:449716 CAPLUS
DN 137:29035
TI Sequences of a human **receptor** for B. **anthracis** toxin and therapeutical uses
IN **Young, John A. T.**; **Bradley, Kenneth A.**; Collier, Robert J.; Mogridge, Jeremy S.
PA Wisconsin Alumni Research Foundation, USA
SO PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002046228	A2	20020613	WO 2001-US30941	20011003

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2000-251481P P 20001205

L12 ANSWER 11 OF 13 CAPLUS COPYRIGHT 2002 ACS
AN 2000:568809 CAPLUS
DN 133:262508
TI Proteolytic activation of **receptor**-bound **anthrax protective antigen** on macrophages promotes its internalization
AU Beauregard, Kathryn E.; **Collier, R. John**; Swanson, Joel A.
CS Department of Microbiology and Molecular Genetics, Harvard Medical School, Boston, MA, 02115, USA
SO Cellular Microbiology (2000), 2(3), 251-258
CODEN: CEMIF5; ISSN: 1462-5814
PB Blackwell Science Ltd.
DT Journal
LA English
RE.CNT 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 12 OF 13 CAPLUS COPYRIGHT 2002 ACS

110067: rakbhat@hotmail.com India
SO Biochemical and Biophysical Research Communications, (September 21, 2001) Vol. 287, No. 2, pp. 542-549. print.
ISSN: 0006-291X.
DT Article
LA English
SL English

L16 ANSWER 40 OF 123 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 2001346234 EMBASE
TI Detoxification of a bacterial toxin by the toxin itself.
AU Montecucco C.
CS C. Montecucco, Dipartimento di Scienze Biomediche, Via G. Colombo n.3, 35121 Padova, Italy. Cesare@civ.bio.unipd.it
SO Trends in Pharmacological Sciences, (1 Oct 2001) 22/10 (493-494). Refs: 7
ISSN: 0165-6147 CODEN: TPHSDY
PUI S 0165-6147(00)01808-3
CY United Kingdom
DT Journal; (Short Survey)
FS 004 Microbiology
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index
LA English
SL English

L16 ANSWER 41 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 11
AN 2001:566770 BIOSIS
DN PREV200100566770
TI Identification of the cellular **receptor** for **anthrax** toxin.
AU Bradley, Kenneth A.; Mogridge, Jeremy; Mourez, Michael; Collier, R. John; Young, John A. T. (1)
CS (1) McArdle Laboratory for Cancer Research, University of Wisconsin-Madison, 1400 University Avenue, Madison, WI, 53706: young@oncology.wisc.edu USA
SO Nature (London), (8 November, 2001) Vol. 414, No. 6860, pp. 225-229. print.
ISSN: 0028-0836.
DT Article
LA English
SL English

L16 ANSWER 42 OF 123 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 12
AN 2001:173763 BIOSIS
DN PREV200100173763
TI Trp 346 and Leu 352 residues in **protective antigen** are required for the expression of **anthrax** lethal toxin activity.
AU Batra, Smriti; Gupta, Pankaj; Chauhan, Vibha; Singh, Aparna; Bhatnagar, Rakesh (1)
CS (1) Centre for Biotechnology, Jawaharlal Nehru University, New Delhi, 110067: rakesh@jnuniv.ernet.in India
SO Biochemical and Biophysical Research Communications, (February 16, 2001) Vol. 281, No. 1, pp. 186-192. print.
ISSN: 0006-291X.
DT Article
LA English
SL English